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RECTAL BLEEDING IN INFANTS AND CHILDREN*

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Bleeding *per rectum* in infants and children may be classified in terms of the quantity of blood passed. Three clinically significant groupings are suggested: (1) slight, less than 1 c.c. in quantity and usually presenting as a drop or so of free blood or as streaking or staining of the stool or of the toilet paper after the stool; (2) moderate, amounting to a dram or so and appearing as free blood mixed or unmixed with the stool; and (3) gross haemorrhage, where the quantity is more than half an ounce at a time and is usually sufficient to give rise to general evidence as loss of blood. Whilst there are likely to be a number of examples in any series of cases where there is no dividing line between these quantitative groupings, yet they serve a useful purpose, and questions on these lines are almost invariably readily appreciated by the parent so that a fairly accurate history can be elicited.

TABLE I. CLASSIFICATION OF RECTAL HAEMORRHAGES: PRE-OPERATIVE DIAGNOSIS AND NUMBER OF CASES

Slight	*Moderate*		*Gross*	
Anal fissure	Polyp	19	Polyp	40
Proctitis:	Multiple polypi	4	Multiple polypi	9
Non-specific	Colitis and proctitis	11	Other tumours	3
Specific	Trauma:		Intussusception	51
Papillitis	Foreign body	2	Colitis and proctitis	4
Cryptitis	Enema nozzle	1	Trauma	3
Prolapse	Cause undiagnosed	18	Peptic ulcer	2
Haemorrhoids			Leukaemia	2
Abscess-in-ano			Thrombocytopenia	2
Fistula-in-ano			Henoch's purpura	1
			Meckel's diverticulum	1
			Cause undiagnosed	26

A review of patients from both hospital and private practice over the 7-year period 1950-56 is analysed in Table I.

SLIGHT HAEMORRHAGE

Most of the cases with slight haemorrhage are of the out-patient class. After attending for one consultation, or occasionally two, they are cured or continue with simple therapy under the care of their family doctors. There are thus no precise comparative figures for this group. However, it is clear that the large majority of slight rectal haemorrhages are due to *anal fissures*. These lesions occur at all ages; they are usually caused by the passage of a hard stool, or sometimes during the course of a bout of diarrhoea, giving rise to the typical, sharp, cutting pain after defaecation, and with a stool commonly described as 'streaked with fresh blood'. Sphincteric spasm is a suggestive sign and frequently prevents throughout examination of the anal verge. In the adult, chronicity of the fissure is common, but in the infant and child it is rare, as the exhibition of mild laxatives or spontaneous softening of the stools for several days together with some lubricant application to the anus, leads to healing.

Proctitis is probably the next commonest cause of slight haemorrhages. It is often found in the course of an attack of diarrhoea or as part of a more general colitis, but many cases occur apart from such conditions. The causes of non-specific proctitis are legion. In children, constipation with faecal impaction is not uncommon, as is also regular purgation administered by over-anxious parents. It is often seasonal and related to new and often not-quite-ripe fruit. On occasion, proctitis has been noted as a complication of acute infections, especially of the oro-pharynx, and it has persisted or even become worse when antibiotics have cleared the original focus. Worm infestation, especially threadworms, commonly causes proctitis. Mucus and pus are often present as well as blood; marked pruritus is a prominent symptom, and tenesmus is common. Sometimes the amount of blood passed is sufficient to put it into the category of 'moderate haemorrhage.'

Under the heading of proctitis, it is convenient to include two localized lesions of the rectum which are occasional causes of slight haemorrhage and produce a clinical picture similar to that of proctitis. The first is *papillitis*, which is being found in increasing numbers in rather older children (10-14 years of age): the second is *cryptitis* which is far less common. Both these conditions with their major distressing symptomatic manifestations, react well to minor operative procedures, and the careful examination required for their diagnosis is invariably well repaid.

Prolapse of the rectum, almost invariably of the partial variety, is an occasional cause of slight or even moderate haemorrhage. It is easily recognized and conservative therapy is usually successful.

Internal haemorrhoids, abscesses, and fistulae-in-ano are all uncommon causes of rectal haemorrhage in children.

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MODERATE AND GROSS HAEMORRHAGE

By contrast with the facility with which a diagnosis of the cause of minor haemorrhage is generally made, the problem in the cases in which bleeding is of more massive character is a major one, because in a remarkably high proportion the cause is not discovered despite the most careful and extensive examination and, sometimes, the cause remains obscure even after exploratory surgery.

It will be noted from Table I that the causes of moderate haemorrhage also operate as causes in the gross haemorrhage category. The essential value of retaining 'moderate' as a distinct class is in relation to the cases itemized as 'Cause undiagnosed'. In general, the quantity of blood passed is the important criterion in assessing the need for laparotomy in the cases in which the cause is not diagnosed, and while this aspect is elaborated below, it is noted here as the justification for classifying 'moderate' haemorrhage as a separate entity.

In our experience, the commonest cause of the more massive forms of haemorrhage is an *adenomatous polyp*. Often palpable on digital examination, usually visible on proctoscopy or sigmoidoscopy and, in a few, revealed by barium enema, the polyp may be extruded during defaecation and its red-ripe raspberry appearance is diagnostic. Its treatment is removal, either *via* the rectum or by laparotomy and colotomy.

The incidence of *multiple polypi*, 13 out of 72 cases of rectal polyp, is a measure of the importance of looking further before accepting the diagnosis of a solitary polyp. The opportunity of a final careful search by sigmoidoscopy during the anaesthesia induced for the removal of the known lesion should not be missed.

Second, and of almost equal frequency as a cause, is *intussusception*. Here, even in the cases where haemorrhage per rectum is the presenting and herald feature, the character of the blood and the clinical picture are usually diagnostic. The occasional atypical, so-called 'chronic,' intussusception may be difficult to recognize, and a barium enema is helpful in settling the matter.

Next in this series come *infections*—enteritis, colitis and proctitis. It is probable that infections are responsible for a much higher proportion of cases or rectal haemorrhage, because many of them do not come to the surgeon but remain with the physician as the haemorrhage is but an incident in the general picture of the enteritis or colitis. Occasionally a *chronic ulcerative dysentery* can only be diagnosed on sigmoidoscopy. There are other cases of haemorrhage *per rectum* which do not present to the surgeon, and the same consideration applies in that their causes should figure more prominently than is reflected in Table I. Such conditions are peptic ulcer, leukaemia, the purpuras, and the haemorrhagic diseases of the newborn.

In peptic ulcer, symptoms referable to the upper abdomen, the occurrence of melaena and an associated haematemesis on occasion, and barium-meal radiography usually lead to, and establish, the diagnosis. However the diagnosis is not invariably simple and straightforward, as is demonstrated by the example of a case in this series where the ulcer was clinically silent, the barium meal was negative, and the sole feature was the melaena stool.

Amongst the cases due to *trauma*, one remarkable case warrants a more detailed note (Fig. 1):

K.V., a boy of 5 years of age, had fallen over veranda pailings on to some recently pruned bushes. A branch of one of these penetrated the skin of his left thigh some 2 or 3 inches above the medial femoral condyle. The boy's father pulled out the broken branch and dressed the small skin laceration. The boy felt bilious soon afterwards and complained of pain; but this did not appear to be severe and medical advice was not sought until some 16 hours after the injury, when an ounce or so of dark blood was passed *per rectum*. This was repeated over the next few hours, the abdominal pain became severe, and vomiting began again. The main features on examination about 30 hours after the accident were the following: Tenderness and rigidity over the hypogastrium; tenderness along the inner side of the left thigh from a small half-inch lacerated wound of the skin at the lower third up to the perineum; marked tenderness and a feeling of bogginess on digital examination of the rectum; and the presence of red blood in the rectum.

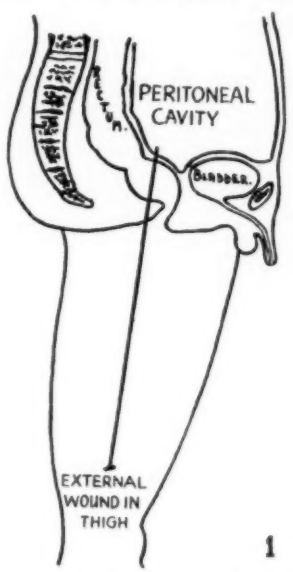

Fig. 1. Case K.V. Diagram of wound track with rectal injury.

Under anaesthesia, proctoscopy showed a laceration of the rectal wall to the left of the mid-line posteriorly, about 2 inches above the anus and, an inch or so higher up, another ragged tear in the anterior rectal wall.

At laparotomy, a pelvic peritonitis with a pool of blood and faecal matter in the recto-vesical pouch was found. The anterior wall of the rectum was perforated just above the peritoneal reflection. This was repaired, the peritoneum was cleaned out and drained, and a colostomy was done. The peri-rectal space, particularly behind the lowest portion of the rectum, was drained by enlarging the track of the penetrating wound and by incising the skin over the track in the perineum.

The subsequent progress was reasonably smooth, and the colostomy was closed after 2 months.

This case was remarkable for the delay in the onset of severe symptoms, and for the rather unusual course of the impaling injury, lending emphasis to the dictum that the diagnosis and treatment of injuries of the rectum are always individual, and usually complex, problems.

The last items in the 2 columns under 'moderate' and 'gross' haemorrhages, labelled 'Cause Undiagnosed' (Table I), represent more than 1/5th of the total number of cases. Investigations including blood counts and blood studies, X-ray screening and proctoscopy and sigmoidoscopy failed to lead to a diagnosis, and a decision had to be made whether it was advisable to seek the cause by exploratory operation.

Apart from the presence of additional features such as an abdominal mass, which may occasionally call for surgical exploration, the outstanding criterion in such a consideration is the severity of the bleeding. On this basis not one of the 18 cases of undiagnosed cause in the 'moderate' group was submitted to operation; in fact during the course of follow-up examinations and repeated laboratory and X-ray investigations, a number of them ceased attending, presumably because the haemorrhage had stopped recurring, and in others such cessation of haemorrhage was confirmed over a lengthy period of regular review.

In the 'gross haemorrhage' group there were 26 cases whose cause was not diagnosed pre-operatively; 4 of them had had a single massive haemorrhage which was not repeated, and the lack of a diagnosis was accepted without the question of laparotomy being raised. These cases illustrate the policy of regarding the primary and real aim of operation as therapeutic in cases not amenable to other forms of treatment and not brought under control by the lapse of time and strictly relegating diagnosis by operation to the position of a means to a therapeutic end. There were 8 instances in this group of unknown cause in which blood transfusion adequately coped with the haemorrhages and their recurrences, and in which, apart from the haemorrhage, there were no other symptoms of the condition; 5 of these cases have stopped bleeding and have been free of it for over 2 years; the other 3 cases are still under observation and the question of surgical intervention is reconsidered from time to time.

Table II presents a summary of the cases exhibiting moderate and gross haemorrhages of unknown cause and their subsequent progress.

TABLE II. PROGRESS OF 44 CASES OF UNKNOWN CAUSE

	Moderate	*Gross*
Solitary haemorrhage	8	4
Spontaneous 'cure' after 1 or more recurrences	10	5
Control by symptomatic treatment (blood transfusion) and still under observation	—	3
Submitted to operation	—	14

The findings in the 14 cases of unknown cause submitted to exploratory surgery are listed in Table III.

TABLE III. FINDINGS AT OPERATION ON 14 UNDIAGNOSED CASES

Polyp of colon	2
Meckel's diverticulum	2
Sarcoma of small bowel	1
Gastric ulcer	1
Contusion injury of sigmoid colon	1
Adenoma and intussusception of jejunum	1
Presence of abnormal vein	1
Cause not established	5

It seems worth remarking on the absence of a case of reduplication of the bowel as a cause of haemorrhage, and this seems to negate the view expressed by Donovan,[1] who places this condition high in importance as a cause of gross intestinal haemorrhage in infants and children.

There are a number of other features of the operative findings that require comment and elaboration.

The 2 cases of Meckel's diverticulum listed in Table III and the 1 case in Table I were heralded by massive haemorrhages associated with the clinical features of sudden and severe anaemia, but they were otherwise symptomless. One case was diagnosed pre-operatively, because the presence of an enterostoma of the umbilicus suggested this diagnosis as the most probable cause of the bleeding. The other 2 cases were discovered at laparotomy; although the presence of a Meckel's diverticulum is strongly suspect as the cause of a symptomless and massive haemorrhage, there is no means of diagnosing it with reasonable certainty except at laparotomy.

The case of haemorrhage due to gastric ulcer is that referred to above as being symptomless and not discovered on X-ray screening.

The contusion injury of the sigmoid colon presented in a child of 2 years of age. No history of the injury was elicited, nor could post-operative enquiries establish the traumatic episode. However, the lesion found at laparotomy seemed to fit this diagnosis better than any other.

No fewer than 5 cases eluded diagnosis despite a careful examination at laparotomy, nor could their causes be found during the subsequent follow-up period: 3 failed to attend for further appointments after a few months; the other 2 reported cessation of the rectal haemorrhage after 4 and 8 months respectively.

The last 2 diagnosed cases listed in Table III, both presented unusual features which, because of their possible bearing on the diagnostic problem as a whole, appear to be worth recording in detail.

B.K., a European girl aged 10 years first presented in June 1951 as a possible acute abdominal condition: and again with a similar clinical picture in January 1952. On both occasions, the symptoms cleared entirely after 48 hours. Investigations afterwards included a barium meal, which was negative. In May 1952 a further bout of abdominal pain, vomiting and, on this occasion, localizing symptoms and signs in the right iliac fossa, led to appendicectomy; however, the appendix is recorded as being of 'doubtful pathology'. Three weeks after this operation she had an attack of severe colic, general in its disposition but most severe in the upper abdomen; and repeated profuse vomiting; she also induced vomiting to relieve the colic. Physical signs were absent.

The symptoms subsided by the 3rd day, and because of the associated constipation, a glycerine and saline enema was given. It produced a stool mixed with dark red blood. This was followed by rapid deterioration of the general condition of the patient, and the signs of peripheral failure became marked, with all the indications of a severe loss of blood. The abdomen became somewhat distended, and tenderness was found in the left hypochondrium. Three further stools consisting of dark red blood, but not black or tarry, were passed in the next hour.

At laparotomy, an extensive irreducible entero-enteric intussusception of the jejunum was found; the whole affected loop of bowel had undergone a volvulus of about three-quarters of a circle, and this loop was non-viable, much of it being frankly gangrenous. The length of affected bowel was resected and an anastamosis was done to re-establish continuity.

The specimen was found to consist of some 7 feet of small bowel; the apex of the intussusceptum was topped by an adenoma (probably benign, but the histological appearances were clouded by the advanced strangulating lesion so that a complete and definite diagnosis remained in doubt).

Her convalescence was surprisingly smooth. Subsequent regular bi-annual examinations, including liver function tests and assessment of protein balance, have been negative.

A feature of considerable interest in relation to the general diagnostic problem, is that relating to the character of the blood passed *per rectum*. Blood originating in appreciable quantity in the small bowel undergoes alteration and can be expected to present as tarry or melaena stools. In this patient this did not occur, and it seems to be important to draw the lesson of such an unusual event: it is essential to include the upper reaches of the small bowel and also the stomach in the examination during laparotomy for the undiagnosed rectal haemorrhage, whatever the character of the blood passed.

The other case, S.M., aged 18 months, recorded in Table III as 'presence of abnormal vein,' is remarkable on account of the considerable rarity of the cause of her rectal haemorrhage (Fig. 2).

Over a period of 6 months, this infant had had 3 massive haemorrhages *per rectum*. On each occasion the blood, bright red in colour, fluid in consistency, and mixed with faecal material, was lost in sufficient quantity to call for urgent hospitalization and the administration of blood transfusions to overcome the obviously

severe exsanguination. On each occasion full investigation, including blood studies, radiological screening, and sigmoidoscopy, failed to produce a diagnosis. Finally, after recovery from the effects of her third haemorrhage, she was submitted to laparotomy.

A large vein (Fig. 2) was found at the base of the mesentery of the sigmoid colon, extending from just above the peritoneal fold at the pelvic floor to near the junction of the left renal vein with the inferior vena cava, both of which latter veins were apparently normal. The abnormal vein, about 2½ inches in length, had a diameter of approximately ½ inch (somewhat more than that of the inferior vena cava) for most of its length. It began by the confluence of two large tributaries in the region of the upper third of the rectum; about ¾ inch proximal to its commencement, there was a communication by a short vertical channel of large

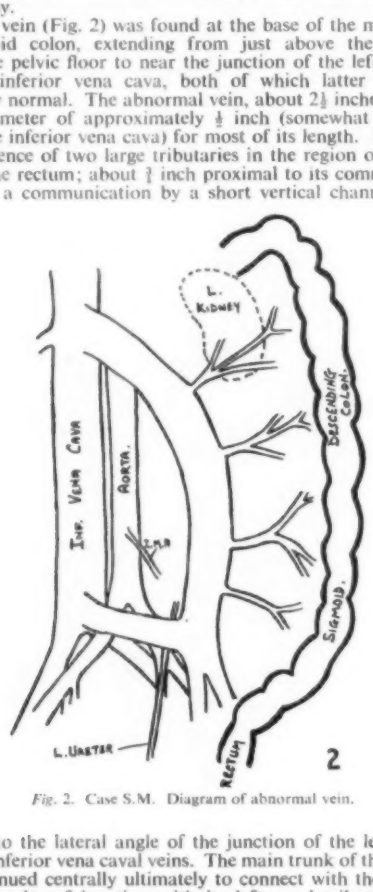

Fig. 2. Case S.M. Diagram of abnormal vein.

diameter to the lateral angle of the junction of the left common iliac and inferior vena caval veins. The main trunk of the abnormal vein continued centrally ultimately to connect with the vena cava just at the site of junction with its left renal tributary. Large radicals from the related portion of the left colon drained into the abnormal vein; a normal inferior mesenteric vein could not be found. The inferior mesenteric artery was of usual anatomical arrangement; it crossed in front of the short communicating venous channel to the commencement of the inferior vena cava. The left ureter was not visualized in the course of dissection and mobilization of the abnormal vein in its upper portion, and it was presumed to be well posterior to this part of the vein; at the lower end, the ureter passed behind the short communicating vein, and then in front of the iliac vessels.

The colon, over a length of approximately 3 inches, from about the apex of the sigmoid loop to the upper portion of the rectum, was congested and bluish in colour and somewhat more thickly walled than the adjacent and paler portions. It was apparent that the portion of the bowel affected by the venous congestion was the site of the haemorrhage which had been passed *per rectum*; and it also seemed highly probable that the abnormal vein was the essential cause of the trouble. Temporary occlusion of the extremities of the abnormal vein demonstrated that the left colon from 2 inches distal to the splenic flexure as far as 1 inch above the pelvic peritoneal reflection was affected. Accordingly this length of bowel was resected (by Paul Mickulicz method) together with the abnormal vein.

A review of the literature, and especially the articles by McClure and Butler,[2] and Huntington and McClure,[3] on the subject of abnormal abdominal veins suggests that the abnormality found in this case is a persistence of the left post-cardinal vein—an abnormality not described in the records available for review.

Apart from the interest in the rarity of this abnormality, the case also points to the necessity for the careful appraisement of the vascular pattern of the bowel in cases of undiagnosed rectal haemorrhage.

SUMMARY

Rectal haemorrhage in infants and children is classified into 3 groups according to the quantity of blood passed. Slight haemorrhage, of up to a few drops at a time, is caused by minor and transient conditions, such as anal fissure, proctitis, anal prolapse, papillitis and cryptitis. Moderate and gross haemorrhages are due to a variety of causes; and the cases over a 7-year period, 1950-56, are tabulated and analysed. A case of trauma to the rectum is presented in detail to give point to the dictum that these injuries are invariably individual problems.

In a remarkably high proportion of cases, despite extensive investigation, including blood studies, sigmoidoscopy and X-ray examination, the cause cannot be diagnosed, and laparotomy must be considered. The decision to operate is based mainly on the quantity of blood lost, whether this is a single episode or whether it is repeated. It is in the light of this criterion for laparotomy that the justification for the classification of the grosser forms of haemorrhage into the 2 categories, 'moderate' and 'gross' is to be found.

The progress of all undiagnosed cases is analysed, and the features of those cases submitted to operation are discussed.

One case of massive haemorrhage due to an adenoma of the jejunum, complicated by intussusception and volvulus, is described in detail, because of the intrinsic interest in the case and also because of the lesson to be drawn from the fact that the haemorrhage *per rectum* presented unchanged, and not as melaena.

Another case, in which the cause of massive haemorrhage in a child of 1½ years of age, was found to be an abnormal vein, probably a persistent post-cardinal vein, is also noted in detail because of the rarity of this abnormality and also because of the possible wider clinical implications of this finding.

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Wes-Kaaplandse Tak: Jaarlikse Gesellige Byeenkoms. Die byeenkoms sal hierdie jaar die vorm aanneem van 'n dinee wat op Vrydag 4 Julie, in die Arthur's Seat Hotel, Seepunt, gehou sal word. Die eggenote en eggenotes van lede is ook welkom. Lede word vriendelik versoek om hul kaartjies so vroeg moontlik by die Takkantoor, Mediese Huis, Waalstraat 35, Kaapstad (telefoon 3-1574, Posbus 643) te kry.

Cape Western Branch: Annual Social Function. The function this year will take the form of a Dinner to be held at the Arthur's Seat Hotel, Sea Point, on Friday 4 July. Members' wives or husbands are welcome to attend. Members are requested kindly to obtain their tickets at the Branch Office, Medical House, 35 Wale Street, Cape Town (telephone 3-1574, P.O. Box 643) at the earliest opportunity.

Suid-Afrikaanse Tydskrif vir Geneeskunde: South African Medical Journal

VAN DIE REDAKSIE

KERLEY SE B-LYNE

Die sukses waarmee myterklep-operasies bekroon is, het 'n nuwe veld in hartnavorsing oopgestel, en in die afgelope 10 jaar is baie metodes uitgewerk en veel ondervinding opgedoen aangaande die bepaling van die geskiktheid van rumatiese pasiënte vir snykundige behandeling. Met behulp van hartkateters is opgawes van die bloeddruk binne die hart en groot bloedvate verkry; opgawes wat die radioloë in verband probeer bring het met die voorkoms van die longgebiede en die hart op hul plate van die borskas. Splinternuwe maatstawwe vir die berekening van die pasiënt se ‚opereerbaarheid' wat nog steeds deur verdere ondervinding gewysig word, is deur hierdie metode bereik. Een van hierdie ‚nuwe' tekens—horisontale en periferale lyne aan die longbodems—is vir die eerste maal in 1951[1] beskrywe deur die Londense radioloog, Peter Kerley, as 'n groep sekondêre strepe in die longgebiede, en hulle het sedertdien bekend geword as ‚Kerley se B-lyne'.

Kerley het gemeen dat hierdie strepe, wat later weer vervaag, limfkanale is waarvan die grootte bepaal word deur die mate van druk by die aar-end van die long-haarbloedvat. Gough[2] skryf die strepe toe aan die ophoping van weefselvloeistof in die tussenwande in die longblasies. Verskeie stukke navorsingswerk is reeds gedoen oor hoe hulle in verband staan met die druk binne die longe—aan die aar-end van die haarbloedvat (linkerkantse voorkamerdruk) aan die een kant, en die slagaar-end (longslagaardruk) aan die ander kant. Rossall en Gunning[3] het 100 gevalle van myterklepvernouing ondersoek, sowel as 16 verdere gevalle van drukverhoging in die longslagaar weens ander oorsake. Die gemiddelde linkervoorkamerdruk is volgens Allison en Linden[4] se metode gemeet; die gemiddelde slagaardruk in die longe is ook bereken—by wyse van bronchoskopie by die meeste van die myterklepgevalle en met behulp van hartkateters by die 16 gevalle van drukverhoging. Die teenwoordigheid van die dwarsstrepe en hul graad is onafhanklik bepaal deur 'n radioloog volgens 'n plaat wat binne 'n maand ná die drukopgawes vasgestel is, geneem is. Die uitslae het 'n duidelike verband tussen die teenwoordigheid van die lyne en 'n verhoogde kapillêre aardruk (linkervoorkamer) aangetoon—hoe hoër die druk, hoe skerper was die lyne afgebeeld. Rossall en Gunning glo inderdaad dat hierdie lyne altyd teenwoordig is wanneer die druk hoër as 24 mm. Hg is, d.w.s. meer as die kolloïed osmotiese druk van die plasmaproteïene (25 mm. Hg), wanneer vloeistof geneig is om in die tussenwande in die longblasies op te hoop en duidelik op X-straalplate afgebeeld word.[2] By die groep myterklepstenose-gevalle was daar nie so 'n duidelike korrelasie tussen die strepe en die kapillêre slagaardruk (longslagaar) nie, hoewel hulle altyd teenwoordig was waar die gemiddelde druk hoër as omtrent 50 mm. Hg was. Hierdie bevinding klop blykbaar met dié van die derde korrelasie, nl. tussen die strepe en longslagaardruk by die 16 gevalle wat slegs drukverhoging in die

EDITORIAL

KERLEY'S B LINES

The success of mitral valvotomy has uncovered a new field of cardiological research, and in the last 10 years many techniques have been developed and much experience gained in assessing the suitability of rheumatic patients for operation. Cardiac catheterization has contributed readings of the blood pressures within the heart and the great vessels, which radiologists have attempted to correlate with the appearances of the lung fields and the heart on their chest films. By this method novel criteria of operability have been reached that are still undergoing modification by experience. One of these 'new' signs—horizontal and peripherally placed lines at the lung bases—was first described by the London radiologist, Peter Kerley, in 1951[1] as a group of secondary lines in the lung fields, and they have since become known as 'Kerley's B lines'.

Kerley held that these transient lines are dilated lymphatic channels whose size depends on the degree of pressure at the venous end of the pulmonary capillary. Gough[2] has ascribed their presence to the accumulation of tissue fluid in the interalveolar septa of the lungs. The way in which they are related to the pulmonary pressures—to the venous end of the capillary (left auricular pressure) on the one hand, and to the arterial end (pulmonary arterial pressure) on the other—has been the subject of several investigations. Rossall and Gunning[3] examined 100 cases of mitral stenosis and 16 more cases of pulmonary hypertension arising from other causes. Mean left auricular pressures were measured by the method of Allison and Linden,[4] and also the mean pulmonary arterial pressure—in most of the mitral cases by bronchoscopy and in the 16 hypertensive cases by cardiac catheterization. The presence of the transverse lines and their degree was assessed independently by a radiologist from a film, usually one which was taken within a month of the pressure recordings. The results showed a clear correlation between the presence of lines and a raised capillary venous (left auricular) pressure—the higher the pressure the more marked the lines. In fact, Rossall and Gunning believe that the lines are invariably present if the pressure exceeds 24 mm. Hg, i.e. a level exceeding the colloid osmotic pressure of the plasma proteins (25 mm. Hg), when fluid tends to collect in the interalveolar septa and produce distinct radiological shadows.[2] Correlation between the lines and the capillary arterial (pulmonary artery) pressure was not so clear cut in the group of mitral stenotics, although above a mean pressure of about 50 mm. Hg they were constantly present. This finding appears to fit the conclusions of the third correlation, i.e. between the lines and pulmonary arterial pressure in the 16 cases showing pulmonary hypertension alone. In only 2 cases were doubtful lines present; in the remaining 14 the films were clear. It seems, therefore,

longe getoon het. Daar was slegs 2 gevalle waar die lyne twyfelagtig was; by die ander 14 was die plate duidelik. Dit blyk dus dat die teenwoordigheid van die lyne en hul graad van intensiteit afhang van die abnormale drukhoogte aan die linkerkant van die hart. Gunning en Rossall beskrywe hul vervlietende teenwoordigheid by 'n geval van linkerhartkamerversaking (wat volg op hartspier-infarksie) en vestig die aandag daarop dat ‚skerp' lyne (in teenstelling met ‚dowwe' of ‚middelmatige' lyne) blykbaar 'n volgehoue hoë linker-voorkamerdruk verg; iets wat by myterklepvernouing wel voorkom, maar nie dikwels by ander aandoenings nie.

Ander navorsers het tot min of meer dieselfde slotsom gekom aangaande die etiologie van hierdie lyne. Bruwer *et al.*,[5] wat hul verlaat het op drukopgawes verkry met hartkateters, het bevind dat die kapillêre aardruk die belangrikste faktor by hul veroorsaking is, en onlangs het Fleming en Simon[6] verslag gedoen oor 99 kateter-korrelasies by 88 pasiënte. Hoewel hul opsomming nie so uitgesproke en afdoende is as dié van Gunning en Rossall nie, weerspreek hulle mekaar in elk geval nie, en hul referaat is uiters belangrik insoverre dit die aandag vestig op onjuisthede of wanbegrippe insake die korreleer van radiologiese en hemodinamiese gegewens. Metode en liggaamshouding speel bv. 'n rol, asook die tussenpose tussen die drukopname en die neem van die borsplaat—hulle meen dat hierdie faktor daarvoor verantwoordelik was dat hulle nie 'n drukmaatstafsyfer kon bepaal waarbó die lyne altyd teenwoordig is nie. Al hierdie werkers stem egter saam dat wanneer Kerley se B-lyne teenwoordig is, die longaardruk hoog is en die graad van myterklepvernouing ernstig genoeg is om drastiese behandeling te regverdig—Gunning en Rossall beweer dat die druk ‚gevaarlik hoog is; . . . dit verdien vroeë oorweging van snykundige behandeling van die klep.'

that the presence and degree of intensity of the lines depends on the abnormal height of the venous pressure on the left side of the heart. Gunning and Rossall describe their transient presence in a case of left ventricular failure (following a myocardial infarct) and point out that 'marked' lines (as contrasted with 'poor' or 'moderate') appear to require a sustained high left auricular pressure, such as is unusual in disorders other than mitral disease.

Other workers have reached more or less the same conclusions on the aetiology of these lines. Bruwer *et al.*[5] relying upon pressures obtained at cardiac catheterization, found the capillary venous pressure to be the more important factor in their production, and recently Fleming and Simon[6] reported upon 99 catheterization-correlations in 88 patients. Whilst their conclusions are altogether more guarded than those of Gunning and Rossall, they are not contradictory, and their paper is invaluable in pointing out the fallacies of correlating radiological and haemodynamic data. Technique and posture play a part, for instance, and also the interval between the pressure readings and the chest film—a factor they believed responsible for their inability to find a critical numerical level of pressure above which the lines are always present. However, all these workers agree that when Kerley's B lines are present, the pulmonary venous pressure is high and the degree of mitral stenosis severe enough to warrant rigorous treatment—in the words of Gunning and Rossall, the pressure is 'dangerously high, warranting early consideration of . . . valvotomy'.

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4. Allison, P. R. and Linden, R. (1955): *Ibid.*, **1**, 9.
5. Bruwer, A. J., Ellis, F. J. and Kirton, J. W. (1955): *Circulation*, **12**, 807.
6. Fleming, P. R. and Simon, M. (1958): J. Fac. Radiol., **9**, 33.

VAGINAALUITSTRYKE IN NORMALE EN VERLENGDE SWANGERSKAP

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Beskrywing van vaginaaluitstryke gedurende swangerskap het reeds van tyd tot tyd sedert 1925 in die literatuur verskyn. Papanicolaou[1] het o.a. in sy beskrywing van uitstryke, wat kenmerkend vir swangerskap is, nadruk gelê op die oorwig van tipiese navikulêre selle uit die intermediêre lae, die groot mate van saamklonting van hierdie selle, die oorvloed van Döderleinse basille en die veelvuldige voorkoms van sitolitiese veranderings deur die Döderleinse organisme veroorsaak. Die beskrywings van Ramirez[2] het grotendeels met dié van Papanicolaou ooreengekom.

Murray[3] was die eerste wat differensiële seltellings gebruik het in die beoordeling van vaginaaluitstryke en het gewys op die toename van selle uit die stratum spinosum superficiale gedurende swangerskap met verminderde wisseling in die kurwes van sy grafieke. Nieburg[4] het ook 'n groot toename van selle uit die intermediêre lae gevind en gemeld dat ná die 30ste week van swangerskap hierdie ‚luteale' selle byna uitsluitlik in die uitstryke aangetref word.

Bonime[5] het 'n verslag gegee van die morfologiese kenmerke wat afgegiete selle gedurende swangerskap onderskei van selle wat gedurende periodes van minder fisiologiese aktiwiteit deur die vaginale slymvlies afgestoot word. Hierdie skrywer het beklemtoon dat eersgenoemde selle, onder invloed van die vermeerderde hormonale produksie gedurende swangerskap, in groter hoeveelhede afgestoot word terwyl hulle nog 'n goedbewaarde en vars voorkoms het, terwyl die selle gedurende periodes van involusie of laer hormonale vlakke gewoonlik 'n ‚ouer,' ingekrimpte en meer verflenterde voorkoms toon met swakgekleurde sitoplasma.

Hopman[6] het o.a. die aandag gevestig op die tipiese groepering van selle wat in die uitstryke van normale swangerskap gevind word. Hierdie groepe toon 'n wolkagtige voorkoms met dikwels 'n waasagtige pers verkleuring in die middel.

Koller en Artner[7] en Wied[8] het indelings van die uitstryktipes wat gedurende normale swangerskap aangetref word, gepubliseer: hulle bevindings het grotendeels ooreengestem. In Tabel I word die bevindings van genoemde skrywers vergelyk met 'n indeling van 589 uitstryke wat op verskillende tydstippe van swangerskap van 126 verskillende pasiënte in die Groningse Vroue-Kliniek[9] verkry is.

In die kornifikasie tipe bestaan die selle grotendeels uit betreklik groot oppervlakte-laagselle waarvan die meeder-

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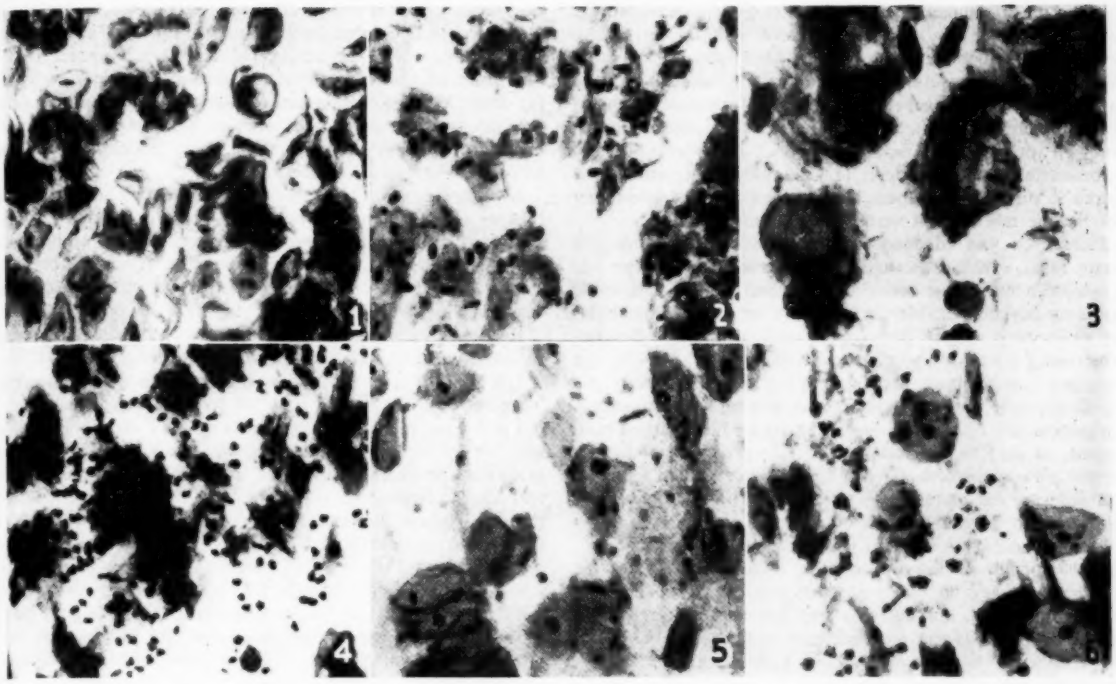

Fig. 1. Vaginaaluitstryk van 'n geval van normale ongestoorde swangerskap van 34 weke, bestaande uitsluitlik uit navikulêre selle met goedgekleurde sitoplasma en duidelike selrande.

Fig. 2. Normale swangerskap 38 weke, die tipiese saamklonting is duidelik sigbaar.

Fig. 3. Sitolitiese tipe smeer soos dikwels in normale swangerskap aangetref word.

Fig. 4. Vaginaaluitstryk 4 dae voor die begin van normale kraam. Toon verminderde saamklonting, onduidelike selrande en wit-bloedselle.

Fig. 5. Vaginaaluitstryk gedurende kraam verkry. Rooibloedselle en fibrine is sigbaar, wit-bloedselle toegeneem, saamklonting verminder, sitoplasma is swak gekleur, selrand is onduidelik.

Fig. 6. Vaginaaluitstryk by 'n geval van vermeende verlengde swangerskap waarby induksie geslaag het. Regressiefaktor 5. (Verminderde saamklonting, wit-bloedselle, slym, onduidelike sitoplasma, onduidelike selrand.)

TABEL I. INDELING VAN UITSTRYKTIPES GEDURENDE NORMALE SWANGERSKAP VOLGENS KOLLER EN ARTNER[7] EN WIED[8] VERGELYK MET BEVINDINGS IN DIE GRONINGSE KLINIEK

Tipe	*Koller en Artner*	*Wied*	*Eie Series*
Kornifikasie tipe ..	2·9%	2·5%	1·2%
Pre-kornifikasie tipe ..	13·1%	10·0%	9·3%
Navikulêre tipe	63·4%	65·0%	56·7%
Sitoliese tipe	15·6%	15·0%	18·0%
Infektiewe tipe	5·0%	7·5%	14·7%

heid gekornifiseer is. Piknotiese kerne oorheers in hierdie tipe. Die meederheid van selle in die prekornifikasie tipe is ook uit die oppervlakte-lae en terwyl die kerne ook grotendeels piknoties is, is die selle hoofsaaklik blou gekleur. In die navikulêre tipe uitstryk bestaan die selle byna uitsluitlik uit ovaal of bootvormige intermediêre laag-selle wat in groepe voorkom. Die kerne is vesikulêr.

Die sitolitiese smere vertoon groot hoeveelhede van los blougekleurde stukkies sitoplasma en los vesikulêre kerne, sowel as die aanwesigheid van Döderleinse organismes.

In die infektiewe tipe uitstryk word 'n groot mate van pseudo-eosinofilie aangetref, die selmembrane is onduidelik, die kerne is grotendeels vesikulêr en perinuklière halos word hier en daar opgemerk. Behalwe hierdie eienskappe van die selle vertoon 1g. uitstryke ook groot hoeveelhede wit-bloedselle en slym. Dit word veronderstel dat die verskillende sitologiese tipes gedurende die swangerskap verband hou met verskille in die verhouding tussen oestrogeen- en progesteroon-invloed.

Gaudefroy,[10, 11] Muller *et al*,[12] Pundel en van Meensel[13] en ander het eosinofiele en karyopiknotiese indices bepaal in die uitstryke van swanger vrouens en het nagegaan watter waardes in ooreenstemming met 'n normale swangerskap bepaal kan word. Die eosinofiele indeks is 'n uitdrukking van die persentasie eosinofiele selle terwyl die karyopiknotiese indeks die persentasie selle met piknotiese kerne weergee. Gaudefroy het 'n eosinofiele indeks van 2 of minder en 'n karyopiknotiese indeks van 30 of minder by ongestoorde swangerskappe ná die 4de maand gevind.

In die reeks van normale gevalle in die Groningse kliniek ondersoek, het die eosinofiele indeks met min uitsonderings onder 20 gedurende die eerste en onder 6 gedurende die laaste twee trimesters gebly terwyl die karyopiknotiese indeks met min uitsonderings onder 20 gebly het.

Aangesien die tipiese swangerskap-uitstryke eers reël-

matig in die 3de maand verkry word, het die vaginaaluitstryke van min waarde by die diagnose van vroeë swangerskap geblyk. Verskillende ondersoekers het hulle egter besig gehou met die kolpositologiese aspekte van die laaste gedeelte van swangerskap en gepaardgaande met verlengde swangerskap. In hierdie verband het vaginaalsmere van meer diagnostiese en prognostiese waarde geblyk. Papanicolaou[1] het veronderstel dat die naderende kraam deur bepaalde kenmerke in die vaginale smeer aangetoon kon word, maar het oor hierdie teorie nooit verder uitgewy nie.

Pundel en van Meensel[13] het 20 gevalle gedurende die laaste fases van die swangerskap waargeneem en kon nie eenvormige sitologiese beelde kenmerkend van die naderende bevalling herken nie; hierdie skrywers het egter gemeld dat die onderwerp verder ondersoek verdien. Labat[14] het opgemerk dat kort voor en gedurende die kraam, selle van die buitenste basale laag vermeerder het ten koste van die navikulêre selle, terwyl karyopiknose neiging tot vermeerdering getoon het; 1g. verskynsel het Labat die mees uitgesproke gevind. In gevalle van verlengde kraam gepaardgaande met verminderde plasentêre aktiwiteit, is 'n soortgelyke toename van selle uit die buitenste basale laag met 'n relatiewe vermindering in navikulêre selle deur Labat waargeneem.

Lemberg en Stamm[15] kon geen duidelike veranderings in die eosinofiele en karyopiknotiese indices kort voor die kraam of in gevalle van verlengde kraam, waarneem nie. Hierdie skrywers het egter sekere kenmerke waargeneem wat hulle as tekens van regressie beskou het ten gevolge van 'n veranderde hormonale ewewig kort voor die kraam. Swakgekleurde selle, onduidelik begrensde protoplasma, die aanwesigheid van slym, fibrine-eksudate en rooi-bloedselle, toename in wit-bloedselle, verminderde afstoting en vermindering in navikulêre selle is deur 1g. skrywers as aanduidings van 'n verandering in hormonale ewewig beskou. Deur van hierdie kriteria gebruik te maak het Lemberg en Stamm die uitstryke in 3 groepe verdeel, nl. ‚normaal voortgaande swangerskap,' ‚byna â terme' en ‚â terme'.

Om moontlike kenmerkende sitologiese veranderings kort voor die begin van kraam en by verlengde swangerskap te ondersoek, is in die Groningse kliniek[9] 278 uitstryke gemaak op verskillende tye binne 28 dae voor spontane kraam wat binne 14 dae van die berekende datum volgens die reël van Naegele plaasgevind het. 'n Tweede groep van 25 pasiënte met ongetwyfeld verlengde swangerskap, is afsonderlik beskou. Van 'n derde groep van 76 pasiënte met vermeende verlengde swangerskap, is die uitstryke beoordeel en hier is 'n poging aangewend om deur middel van die sitologiese ondersoek vooraf te bepaal of induksie sou slaag of nie. 'n Geslaagde induksie is beskou as een waarby 'n mediese induksie binne 24 uur kontraksies veroorsaak het wat uiteindelik tot die kraam gelei het.

Die sitologiese bevindings is noukeurig op sitogramme genoteer; 'n differensiële seltelling is gemaak van elke uitstryk; vir hierdie doel is in elke smeer 400 selle getel. Die eosinofiele indeks, die piknotiese indeks, sowel as die kolpositologiese indeks (die verhouding tussen oppervlakteselle en selle uit die dieper lae) is genoteer. Die gemiddelde grootte van die selle en van die kerne is genoteer sowel as die kleurbaarheid en voorkoms van die sitoplasma, die voorkoms van die selmembraan, die mate van groepering van die selle, die mate van opkrul en vou van die selrande, en van sitoliese, die aanwesigheid van slym, wit- en rooi-bloedselle en organismes. Uit die 1g. eienskappe van die uitstryke is gepoog om tussen verskillende grade van regressiewe veranderings te onderskei. Die volgende 9 faktore is beskou as aanduidings van regressie: (1) 'n klein gemiddelde seldeursnee, (2) 'n klein gemiddelde kerndeursnee, (3) swakgekleurde sitoplasma, (4) onduidelike selmembraan, (5) verminderde saamklonting, (6) 'n oormate van opkrul en vou van selrande, (7) vermeerderde slym in die smeer, (8) vermeerde leukosiete, (9) die aanwesigheid van rooi-bloedselle.

By gebrek aan 'n beter benaming is die totaal van hierdie faktore uitgedruk as die regressiefaktor (regression rating). Dit word besef dat hierdie gradering geen rekenskap hou met die relatiewe belangrikheid van die individuele faktore nie, tog is gevind dat die regressiefaktor 'n baie behulpsame weerspieëling was van die hormonale ewewig.

Resultate van die sitologiese bevindings by die bg. groepe pasiënte word in Tabelle II, III en IV verstrek.

Uit Tabel II blyk dat by normale ongestoorde swangerskap 'n eosinofiele indeks van meer as 10 byna altyd daarop dui dat die kraam binne 8 dae verwag kan word. Dieselfde geld vir 'n piknotiese indeks van meer as 20 en vir 'n kolpositologiese indeks van meer as 0.5. Die teenoorgestelde van hierdie bevindings is egter nie geldig nie aangesien baie pasiënte indices gehad het ver onder hierdie grense, kort voor spontane bevalling. Vir die groep pasiënte waarvan die swangerskap tot 42 weke of langer verleng was (Tabel III), het dieselfde gegeld. Indices bo die genoemde grense was sterk aanduidings van beginnende kraam binne 8 dae, maar selfs meer as in die vorige groep was lae indices hier van min betekenis om die datum van beginnende kraam te voorspel, en was geen aanduiding van die verlengde duur van swangerskap nie.

Wat die regressiefaktor betref, hier toon tabelle II en III albei dat 'n regressiefaktor van 3 of meer 'n sterk aanduiding is van die waarskynlikheid dat die kraam binne 8 dae sal begin terwyl 'n regressiefaktor van minder as 3 in hierdie geval 'n meer betroubare aanduiding was dat die swangerskap vir nog meer as 8 dae sal voortgaan. By verlengde swangerskap was die regressiefaktor met één uitsondering 3 of meer.

Van 'n verdere groep van 213 pasiënte met ongestoorde swangerskappe en wat almal spontaan beval het binne 14 dae van die berekende datum volgens Naegele se reël, is van elkeen slegs een uitstryk gemaak. Hierdie uitstryke is gemaak op verskillende tye van 1 dag tot 28 dae voor die kraam. Van 114 van hierdie gevalle met 'n regressiefaktor van 3 of meer het 108 (94.7%) binne 8 dae beval. Van 99 met 'n regressiefaktor van minder as 3 het die swangerskappe in 76 gevalle (76.8%) langer as 8 dae bly voortbestaan, terwyl 23 binne 8 dae beval het. By die 76 gevalle van vermeende verlengde swangerskap waar die duur van die swangerskap vermoedelik 42 weke oorskry het, het mediese induksie geslaag in 38 gevalle en in dieselfde aantal nie geslaag nie. Van die 38 geslaagde gevalle het 35 (92.1%) 'n regressiefaktor van 3 of meer gehad. Van die 38 mislukte gevalle het 5 (13.2%) 'n regressiefaktor van 3 of meer gehad. Die eosinofiele indeks, die piknotiese indeks en die kolpositologiese indeks het hier van minder waarde geblyk (Tabel IV).

Dit word aan die hand gegee dat die beoordeling van vaginaaluitstryke en veral die berekening van die mate van regressie 'n nuttige en praktiese hulpmiddel kan blyk by die diagnose van verlengde swangerskap. Kliniese en radiologiese metodes het vir hierdie doel nie baie betroubaar geblyk nie, terwyl bepalings van hormoonuitskeiding praktiese moeilikhede inhou, en ook dikwels teleurstellende resultate gelewer

TABEL II. SITOLOGIESE INDICES IN 278 UITSTRYKE VERKRY GEDURENDE DIE LAASTE 28 DAE VOOR SPONTANE KRAAM

Duur van swangerskap minder as 42 weke	*EI* 0–2	*EI* 3–5	*EI* 6–10	*EI* 11–20	*EI* 21–50	*EI* >50	*PI* 0–2	*PI* 3–5	*PI* 6–10	*PI* 11–20	*PI* 21–50	*PI* >50	*KI* 0·–·02	*KI* ·03·–·05	*KI* ·06·–·1	*KI* ·11·–·2	*KI* ·21·–·5	*KI* ·51–1	*KI* >1	*RF* 0	*RF* 1	*RF* 2	*RF* 3	*RF* 4	*RF* 5	*RF* 6	*RF* 7	*RF* 8	*RF* 9
Spontane bevalling binne 1–2 dae ..	31	30	30	31	25	7	48	30	27	34	16	2	33	15	15	14	18	18	44	3	3	10	11	24	43	33	24	5	—
Spontane bevalling binne 3–4 dae ..	12	6	4	3	2	—	10	10	2	3	2	—	12	3	3	1	—	6	1	3	2	5	6	7	1	3	—	—	—
Spontane bevalling binne 5–6 dae ..	7	5	2	2	1	—	8	7	1	1	—	—	7	3	2	1	4	—	—	2	3	2	2	2	3	3	—	—	—
Spontane bevalling binne 7–8 dae ..	12	5	2	1	2	—	9	8	2	2	—	—	5	7	5	—	4	—	—	3	1	3	4	2	6	2	—	—	—
Spontane bevalling binne 9–10 dae ..	14	—	—	—	—	—	7	2	—	4	1	—	5	1	2	3	1	—	1	8	3	1	—	1	—	1	—	—	—
Spontane bevalling binne 11–12 dae ..	9	—	—	—	—	—	7	3	—	1	—	—	6	1	2	1	—	—	—	6	1	2	1	—	—	—	—	—	—
Spontane bevalling binne 13–14 dae ..	7	1	1	—	—	—	7	2	—	—	—	—	6	1	—	—	—	—	—	6	1	—	1	—	1	—	—	—	—
Spontane bevalling binne 15–16 dae ..	9	3	—	—	—	—	6	5	—	1	—	—	5	1	3	1	2	—	—	7	3	—	—	1	—	—	1	—	—
Spontane bevalling binne 17–18 dae ..	8	1	1	1	—	—	10	1	1	—	—	—	7	1	—	1	2	—	—	6	1	3	1	—	—	—	—	—	1
Spontane bevalling binne 19–20 dae ..	5	—	—	—	—	—	3	2	—	—	—	—	2	1	2	—	—	—	—	2	1	1	1	—	—	—	—	—	—
Spontane bevalling binne 21–22 dae ..	7	1	—	1	—	—	7	1	1	—	—	—	4	2	1	1	1	—	—	5	2	1	1	—	—	—	—	—	—
Spontane bevalling binne 23–28 dae ..	13	5	1	—	—	—	10	6	2	—	—	—	7	3	2	3	2	1	—	16	2	—	—	—	—	—	—	—	—

Dit blyk dat 'n eosinofiele indeks (EI) hoër as 10 byna altyd 'n bevalling binne 8 dae voorspel, terwyl aan 'n piknotiese indeks (PI) bo 20 en 'n kolpositologiese indeks (KI) bo 0·5 dieselfde betekenis geheg kan word. Die teenoorgestelde is egter by nie een van hierdie indices van toepassing nie. 'n Regressiefaktor (RF) van 3 of meer mag hieruit ook as 'n aanduiding vir kraam binne 8 dae beskou word, terwyl die teenoorgestelde in hierdie geval ook meer juis blyk.

TABEL III. SITOLOGIESE INDICES IN 25 UITSTRYKPREPARATE VAN PASIËNTE BY WIE SWANGERSKAPSDUUR 42 WEKE OORSKRY HET

Duur van swangerskap meer as 42 weke	*EI* 0–2	*EI* 3–5	*EI* 6–10	*EI* 11–20	*EI* 21–50	*EI* >50	*PI* 0–2	*PI* 3–5	*PI* 6–10	*PI* 11–20	*PI* 21–50	*PI* >50	*KI* 0·–·02	*KI* ·03·–·05	*KI* ·06·–·1	*KI* ·11·–·2	*KI* ·21·–·5	*KI* ·51–1	*KI* >1	*RF* 0	*RF* 1	*RF* 2	*RF* 3	*RF* 4	*RF* 5	*RF* 6	*RF* 7	*RF* 8	*RF* 9
Spontane kraam binne 1–2 dae	3	—	4	4	—	—	4	2	3	2	1	—	1	—	2	4	2/7	1	2	—	1	—	—	2	2	5	2	—	—
Spontane kraam binne 3–4 dae	3	1	1	2	1	—	4	1	—	3	—	—	1	—	2	—	4	—	1	—	—	—	1	2	3	2	—	—	—
Spontane kraam binne 5–6 dae	1	—	—	—	—	—	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—
Spontane kraam binne 7–8 dae	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	1	—	—	—	—
Spontane kraam binne 9–10 dae	1	—	1	1	—	—	1	—	1	1	—	—	—	1	—	1	—	1	—	—	—	—	2	—	—	1	—	—	—
Spontane kraam binne 11–20 dae ..	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	1	—	—	—	—	—

Die smere is op verskillende tydstippe van 1-12 dae voor spontane kraam gemaak. Die eosinofiele, piknotiese en kolpositologie se indices het in hierdie groep geen aanduidings gegee van serotiniteit nie. Die regressiefaktor was hoër as 2 in op een na alle gevalle.

TABEL IV. SITOLOGIESE INDICES BY 76 PASIËNTE BY WIE DIE SWANGERSKAPSDUUR VERMOEDELIK 42 WEKE OORSKRY HET EN BY WIE MEDISINALE INLEIDING VAN DIE KRAAM PROBEER IS

	EI 0–2	*EI* 3–5	*EI* 6–10	*EI* 11–20	*EI* 20–50	*EI* >50	*PI* 0–2	*PI* 3–5	*PI* 6–10	*PI* 11–20	*PI* 21–50	*PI* >50	*KI* 0·–·02	*KI* ·03·–·05	*KI* ·06·–·1	*KI* ·11·–·2	*KI* ·21·–·5	*KI* ·51–1	*KI* >1	*RF* 0	*RF* 1	*RF* 2	*RF* 3	*RF* 4	*RF* 5	*RF* 6	*RF* 7	*RF* 8	*RF* 9
Inleiding geslaag	9	8	7	5	9	—	12	6	11	4	4	1	7	2	3	3	11	7	5	0	1	2	5	8	10	9	3	—	—
Inleiding misluk	25	6	4	1	2	—	26	6	6	—	—	—	16	5	6	3	3	3	2	13	9	11	5	—	—	2	—	—	—

Die inleiding is as geslaag geag as kontraksies wat uiteindelik tot die kraam gelei het binne 24 uur begin het nadat die inleiding begin is.

het. By die kies van gevalle vir induksie van die kraam, behoort hierdie metode van waarde te wees.

OPSOMMING

'n Kort oorsig van die literatuur betreffende vaginaaluitstryke gedurende swangerskap, word gegee. Kenmerkend van uitstryke gedurende normale swangerskap verkry, is die groot opeenhopings van navikulêre selle met vesikulêre kerne, goedgekleurde sitoplasne en duidelike selrande en die relatief geringe hoeveelheid slym en wit-bloedselle. In 'n reeks van 589 uitstryke van pasiënte met ongestoorde swangerskappe, was 56.7% van die navikulêre tipe, 18.0% van die sitoliese tipe, 14.7% van die infektiewe tipe, 9.3% van die pre-kornifikasie tipe en 1.2% van die kornifikasie tipe. Uit 278 uitstryke verkry gedurende die laaste 28 dae voor spontane kraam, by 25 pasiënte met ongetwyfeld verlengde swangerskap en by 76 gevalle van vermeende verlengde swangerskap, het geblyk dat die eosinofiele, piknotiese en kolpositologiese indices, hoewel van positiewe waarde om die tydstip van begin van die baring te voorspel en om verlengde swangerskap te konstateer, van geen negatiewe waarde was nie. Die regressiefaktor wat bereken is as die totaal van 9 moontlike kriteria vir die herkenning van regressiewe verskynsels in die uitstryke het 'n meer betroubare indeks geblyk. Van 114 gevalle met 'n regressiefaktor van 3 of meer het 108 binne 8 dae gekraam en 99 met 'n regressiefaktor van minder as 3 het die swangerskappe in 76 gevalle langer as 8 dae bly voortduur.

Bepaling van die regressiefaktor het ook van praktiese waarde geblyk by die kies van gevalle vir induksie by vermeende verlengde swangerskap.

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SIDEROPENIC DYSPHAGIA

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It has been well known for many years that glossitis and dysphagia may occur in association with chronic iron-deficiency anaemia. In Scandinavia the condition has been found to be especially common and cases have been described in which there have been marked changes in the oesophagus due to iron deficiency, but no anaemia.[1, 2] This unusual presentation, however, has been only rarely noted in the English literature,[3] and for this reason the present case is reported in detail.

CASE RECORD

The patient was first seen in June 1957. She was a married female aged 43 years who had come to South Africa from Holland 8 years previously. Her major complaints were difficulty in swallowing and soreness of the tongue, both of which had been present for 20 years. The symptoms had become worse in the preceding 3 months and the dysphagia was particularly troublesome. Both solids and liquids seemed to stick at the level of the cricoid cartilage and the dysphagia was so marked that the patient was forced to spend 1–2 hours over each meal. Vomiting was not a feature but food sometimes regurgitated into the mouth and she often woke at night to find her mouth full of saliva. Although the appetite remained good the dysphagia caused the patient to restrict her diet to soup, milk, occasional vegetables but no meat. On this diet the weight remained stationary for years at 127 lb., but in the few months before examination she had lost 8 lb.

Over the years the patient had consulted several doctors because of the dysphagia. In 1939 a diagnosis of 'nerves' was made; at that time she was told she was anaemic but no blood tests were done and no specific treatment was given. In 1946 her first child was born; she remained well during the pregnancy and there was no excessive postpartum haemorrhage.

In 1953, when she was in a maternity hospital for her second confinement, a hypochromic anaemia was diagnosed (haemoglobin 5·2 g.%). To the patient's knowledge there had never been any abnormal blood loss. Menstruation had always been normal and she had never vomited blood or passed it *per rectum*. In view of the low haemoglobin level she was given 4 pints of blood. At the time of discharge from the maternity hospital the haemoglobin had risen to 13·2 g.% and she received no further treatment.

In February 1956, an ear, nose and throat surgeon reported spasm at the level of the cricopharyngeus muscle on oesophagoscopy and this was said still to be present on barium swallow when she was re-examined in September 1956. The constriction was dilated with temporary relief of the dysphagia for about 2 weeks. Several blood tests were done during 1956; these showed persistently normal values, with haemoglobin levels varying between 13·3 and 16·3 g.%. In spite of this a short course of oral iron was given but the patient noticed no real improvement while on treatment. She was seen again in June 1957 by another physician. The haemoglobin level was 15·4 g.% and she was referred to the Johannesburg General Hospital for further investigation.

On examination she appeared a healthy, well-balanced woman. The main positive features were marked koilonychia and glossitis. The tongue appeared raw and beefy with atrophy of the papillae. Nothing else abnormal was noted on physical examination.

Special Investigations. Haemoglobin 17·0 g.%, haematocrit 52%, white blood count 4,000 per c.mm., film appearances normal. Sedimentation rate (Wintrobe) 3 mm. in 1 hour. Serum iron 93 gamma %, total iron-binding capacity 405 gamma %. Bone-marrow examination showed normoblastic erythropoiesis and no visible haemosiderin granules in unstained marrow particles and in specimens stained with Prussian blue. Plasma proteins 6·6 g.% (albumin 3·1, globulin 3·5). Blood urea 28 mg. per 100 ml. Gastric analysis showed no free acid present in fasting specimens, but after Kay's 'augmented histamine test'[4] (0·4 mg. per kg. given subcutaneously with 50 mg. of intramuscular mepyramine maleate) 13 c.c. of N/10 HCl in 100 c.c. of gastric juice. Absorption of radio-active B12 (measured as the amount excreted in a 24-hour specimen of urine when a 'flushing' dose of 1,000 micrograms was given 2 hours after the radio-active one) 17% (normal range 10–30%). Urine analysis normal. Stool examinations showed no occult blood.

On barium swallow (Fig. 1*a*) a stricture of the upper oesophagus

was shown projecting into the lumen mainly from the anterior wall in the manner of a web. On deglutition the oesophagus above the constriction bulged out to form a pseudo-diverticulum or pouch. In the anterior position, barium from both pyriform fossae was diverted across to the left and the stricture was again shown about 1½ inches below the crico-pharyngeus. For a short distance below this the oesophagus appeared narrowed and slightly irregular.

Technical Note on Radiological Examination. A web may easily be missed on routine examination and specialized technique was used for the barium-swallow examination. The method was to count aloud up to 'five' after having instructed the patient to swallow as large a bolus of barium cream as possible with a single swallowing movement at the count of 'two'. The passage of barium was then observed in order to assess the best time to expose a radiograph of the distended pharynx with the next mouthful. For such a study a rapid-acting spot-filming device is essential and the exposure should be not greater than 1/10 second if a sharp radiographic image is to be obtained.

Progress

Because of the severity of the dysphagia an oesophagoscopy was performed before treatment was begun. There was marked narrowing of the upper oesophagus. Above this the pharyngeal mucosa appeared normal. The constriction was then gently dilated. Although the mucosa felt roughened beyond the constriction no actual neoplasm was visualized. A biopsy specimen was taken from the mucosa distal to the constriction. Histologically the submucosa appeared slightly oedematous, with small round-cell infiltration, but there was no evidence of malignancy. The oesophageal dilatation caused prompt relief of the dysphagia and the patient was able to consume a normal diet without discomfort.

In hospital the iron stores of the body were replenished by a course of intramuscular iron (Imferon), a total of 1,600 mg. being administered. A follow-up barium swallow while the patient was still in hospital showed some improvement (Fig. 1 b). An anterior web was again shown about 1½ inches below the crico-pharyngeal sphincter but the lumen of the oesophagus below it was much broader and more easily seen. A small posterior web was also visualized slightly below the upper one. The oesophagus below the level of the web was demonstrated by a double contrast manoeuvre. It showed narrowing for a short distance but the surface appeared smooth.

Follow-Up

After discharge from hospital the patient was seen regularly at monthly intervals. She remained in good health for 6 months, experiencing no difficulty with swallowing. A repeat barium swallow 3 months after discharge showed that most of the objective improvement had been maintained, although the lumen was slightly narrower than on the previous study (Fig. 1c). Similar findings were made at a further examination in January 1958. The patient had by this time gained 10 lb. in weight and was feeling well. Moreover the tongue was no longer sore and regeneration of papillae could be seen. On odd occasions some dysphagia was experienced, especially with meat, but on the whole she was able to eat a full diet without difficulty. At this check-up another oesophagoscopy was carried out and the stricture was dilated again.

DISCUSSION

The association of iron-deficiency anaemia with glossitis and dysphagia has been described as the Plummer-Vinson syndrome. The condition is especially common in Sweden, where Waldenström and Kjellberg[1] reported its incidence as twice that of oesophageal carcinoma. It was at first thought that the dysphagia was either due to hysterical spasm[5] or to a derangement of the nerve plexuses.[6, 7] However, it has now been established that the dysphagia is associated with organic changes in the oesophagus.[8-10]

On radiological examination the most striking feature is a web which occurs in the upper part of the oesophagus between the level of the cricoid plane and the suprasternal notch. It is seen as a thin membranous shadow extending from the anterior wall of the oesophagus into the barium-distended lumen.[11-15] In severe cases it is more extensive and encircles the oesophageal lumen as a narrow ring structure[15] (Fig. 1). Sometimes a segment of narrowed oesophagus may be seen extending about ½ inch below the web, with occasionally a second web present at the lower limits of the narrowed area.[14] When the web is sufficiently extensive it can be seen in antero-posterior views as a localized narrowing

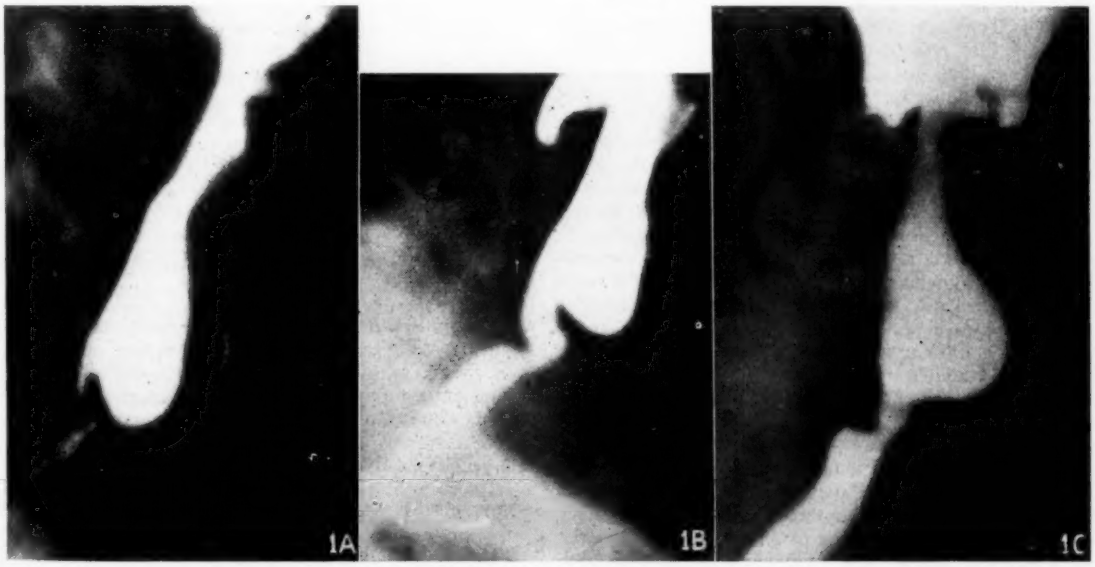

Figure 1*a.* Initial barium swallow, demonstrating the severe web stricture, with oesophageal dilatation above. *Figure* 1*b.* Barium swallow soon after oesophageal dilatation, showing the anterior and posterior webs. *Figure* 1*c.* Upper oesophageal film several months after dilatation. The changes are still present, but not as severe as before therapy.

of the distended oesophagus. If the degree of obstruction is marked, the barium bolus may cause the pharynx and oesophagus above the constriction to bulge out and the appearances may simulate a pouch (Fig. 1a). The differential diagnosis is usually not difficult since the radiological findings are quite unlike those of carcinoma and indentations of the oesophagus caused by anterior cervical osteophytes. The condition most likely to be confused with a web is spasm of the crico-pharyngeus.[19] However, the indentation by the muscle is posterior and is usually broader than a web.

Very little is known of the histological changes found in the Plummer-Vinson syndrome. In a case which came to autopsy Suzman[8] found hyperkeratinization of the oesophageal epithelium with areas of atrophic degeneration, while Hoover[9] reported that a web removed by punch biopsy consisted of two layers of mucous membrane separated by a small amount of fibrous tissue. More recently Savillahti[10] found localized atrophy and chronic inflammatory changes in the epithelium of a case which came to autopsy, and atrophic changes were noted in the muscular coat.

Although there was at first some dispute about the significance of the oesophageal changes found in states of iron deficiency there is no doubt now that they are caused by a depletion of tissue iron. Waldenström,[1] in 1937, was one of the first to stress the fact that iron deficiency can lead to widespread epithelial changes. He considered koilonychia, glossitis, achlorhydria and oesophageal webs as all manifestations of deficiency of tissue iron. He therefore proposed the term 'sideropenic dysphagia' as a rational way of describing difficulties in swallowing due to iron deficiency. In the past 2 decades iron metabolism has been extensively studied and the results have done much to confirm Waldenström's thesis. A consideration of this work is necessary in order to understand the proper significance of sideropenic dysphagia and to formulate a rational therapy.

There are about 4 g. of iron in the body of a normal adult male. The major portion is in the form of haemoglobin, but iron is also present in myoglobin, in the plasma, and in tissue enzymes. In addition, 1-1.5 g. are contained in the stores of the body.[16] Once introduced into the body, the iron is used again and again, with the result that only small amounts are normally absorbed and excreted at any one time. This, however, is less true in the female, where losses are normally incurred through menstruation and pregnancies. When such losses become excessive or when the diet is inadequate the iron balance may become negative. In such circumstances a fixed sequence of events occurs.[17] At first iron is mobilized from the stores to satisfy the needs of the bone marrow. When the stores have been depleted the level of iron in the plasma drops and finally anaemia develops. At first the anaemia is normocytic but, with growing deficiency in available iron, haemoglobin synthesis becomes impaired and a microcytic, hypochromic anaemia develops. It is at this stage that signs of epithelial damage usually develop. However, not all cases with epithelial changes manifest anaemia and although reports of such cases are rare in the English literature[3] they have been well documented by Scandinavian workers.[1, 2] In these cases the iron deficiency is often reflected by the finding of a low plasma-iron level. Metabolically it seems very probable that patients with sideropenic dysphagia and no anaemia are precariously balanced at a point where the stores are totally exhausted but enough iron is available from the breakdown of red-cells to satisfy the needs of the bone-marrow. In this state of delicate equilibrium, haemoglobin synthesis takes top priority, with the result that even the modest needs of the tissues are not met.

The case reported here appears to be an excellent example of such a metabolic upset. At the time she was first seen she had been complaining of symptoms for 20 years. The haemoglobin level was normal, but there was atrophic glossitis, koilonychia and marked dysphagia. Fasting achlorhydria was also present, but some acid secretion was produced with the 'augmented histamine test'[4] and the absorption of vitamin B12 was normal. Although the plasma-iron level was not lowered, no storage iron could be demonstrated in the bone-marrow specimen. It was known that she had suffered from a severe microcytic hypochromic anaemia 3 years previously at the birth of her second child. At that time she was given blood transfusions which restored the haemoglobin level to normal, but were apparently not sufficient to satisfy the tissue needs. The epithelial symptoms therefore continued to progress and 3 years later the glossitis and dysphagia were the presenting symptoms.

In mild cases it has been found that oral iron therapy causes prompt amelioration of the dysphagia. Although subjective relief is usually obtained, the radiological findings may show little change, a fact which suggests that spasm may play a part in the production of symptoms.[14] In severe cases it is usually necessary to dilate the oesophagus before starting treatment with iron. In the past it has been customary to use oral iron. Although such therapy is very effective in returning the haemoglobin level to normal it is extremely difficult to replenish body stores by the oral route.[18] There thus seems to be an excellent case for using parenteral iron in the treatment of the condition. In the present patient the iron-dextran complex, Imferon, was given intramuscularly with good results. It should be emphasised that the changes in the oesophageal epithelium have been shown to be pre-malignant by a number of authors,[1, 10, 13, 14] and the high incidence of hypopharyngeal and upper oesophageal carcinoma in Scandinavian women may well be due to the prevalence of sideropenic dysphagia in this area.[13] All cases *must* therefore be given adequate iron therapy; this means enough to correct anaemia, when present, and to refill the iron stores (i.e. an extra 1,500 mg.). In addition, every patient suffering from sideropenic dysphagia should be examined radiologically at 6-monthly intervals. Any irregularity of the oesophageal lumen above or below the web should be regarded with suspicion and investigated further.

SUMMARY

A case is described where iron deficiency caused marked epithelial changes (glossitis, oesophageal stricture and koilonychia) but no anaemia. Dysphagia, which was the most prominent symptom, was treated by oesophageal dilatation and intramuscular iron therapy with good results.

The diagnosis of sideropenic dysphagia is discussed and the pathogenesis of the condition is briefly outlined.

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CHRONIC IDIOPATHIC THROMBOCYTOPENIA IN THE BANTU

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The literature on idiopathic thrombocytopenia in the Bantu consists largely of papers on 'Onyalai'. The acute nature of the condition has been repeatedly stressed, but the existence of recurrent[1-3] and chronic[4, 5] forms, have been suggested. Manson-Bahr[6] considers that the bullae of Onyalai, once resolved, never recur, and concludes that it is an acute form of thrombocytopenic purpura.

During the course of a study of idiopathic thrombocytopenia in the Bantu, 7 cases regarded as examples of the chronic form, were encountered. The material reviewed, and the methods used, have been described in a previous paper.[7] The 7 comprise 4 cases seen in at least 2 attacks and 3 cases where bleeding and thrombocytopenia have so far persisted for 2 years, 18 months and 10 months respectively. The platelets have failed to return to normal levels, and the bleeding tendency is constant. In view of the fact that few authentic cases have hitherto been described in the Bantu, the cases are briefly reported individually.

CASE REPORTS

Case 1

A.M., a Bantu female aged 27 years, was admitted to hospital in October 1953 with a 2-day history of epistaxis. There had been previous epistaxis 16 months before. Physical examination showed generalized purpura, bleeding gums, and a single haemorrhagic vesicle on the palate. The spleen was not palpable. The patient was 32 weeks pregnant. The haemoglobin was 6·8 g. per 100 ml., leucocytes 10,200 per c.mm. (eosinophils 102 per c.mm.), and platelets 45,000 per c.mm. The marrow contained normal numbers of megakaryocytes, but these showed no evidence of platelet formation.

Blood transfusion was given, and ACTH 100 mg. daily was commenced on the 4th day. Clinical remission coincided with the exhibition of ACTH, but thrombocytopenia persisted (platelets 20,000-30,000 per c.mm.), and the patient was discharged after 1 month, still thrombocytopenic (platelets, 30,000 per c.mm.).

Two months later, with platelets 30,000 per c.mm., she bore a live infant. There was no excessive postpartum haemorrhage, and the infant was not thrombocytopenic and showed no purpura. Relapse, with bleeding from the mouth and purpura, occurred in the mother 2 days after delivery, with platelets 20,000 per c.mm.; the marrow picture was as before. The direct Coomb's test was negative, and 'L.E. cells' were not detected. Haemorrhagic manifestations (purpura, epistaxis, and vaginal bleeding) and thrombocytopenia persisted over the following 3 months, in spite of therapy, including blood transfusion, ACTH 80 mg. daily, and cortisone 100 mg. daily. Splenomegaly was not noted at any stage.

With haemoglobin 6·6 g. per 100 ml. and platelets 75,000 per cu.mm., and in spite of continued haemorrhage, the patient refused splenectomy, and left hospital against medical advice. She died at home one week later—autopsy was not possible. The total observed course in this patient was 6 months.

Case 2

E.N., a Bantu female aged 36, was transferred in March 1955 from an outside hospital, where she had received 2,000 ml. of blood over a period of 2 months for persistent epistaxis. One month before the onset of epistaxis she had borne an infant without excessive haemorrhage, and there was no purpura in the infant.

Physical examination on admission showed bullae in the mouth, generalized purpura, epistaxis, and fundal haemorrhages. The spleen was not palpable. Bleeding time longer than 15 minutes, and tourniquet test negative. Haemoglobin 6·0 g. per 100 ml., leucocytes 6,200 per c.mm. (eosinophils 0%), and platelets 20,000 per c.mm. Megakaryocytes were present in the marrow in normal numbers, but showed no evidence of platelet-budding. Direct Coombs test negative. 2,000 ml. of blood was transfused, and cortisone, 300 mg. daily, was commenced on the 4th day, decreasing over the following week to 75 mg. daily. There was clinical remission, but thrombocytopenia persisted (platelets less than 20,000 per c.mm.), and the patient was discharged 1 month after admission.

In May 1956, 14 months later, she was readmitted to hospital. Four months before she had delivered another infant, with no excessive postpartum haemorrhage. There was no purpura in the infant. A few days after delivery there was epistaxis, bleeding gums, and vaginal bleeding, these symptoms persisting up to the time of admission. Examination showed generalized purpura, numerous bullae in the mouth, and haematuria; the spleen was not palpable. Blood count showed haemoglobin 4·4 g. per 100 ml., leucocytes 6,600 per c.mm. (eosinophils 0%), and platelets less than 20,000 per c.mm. Numerous megakaryocytes were present in the marrow, but they showed cytoplasmic hyalinization, 'lymphoid' forms, and failure to bud. 'L.E. cells' were not detected.

Blood transfusion (1,000 ml.) was given, and 30 mg. of prednisone daily was commenced on the 6th day. Fresh purpura, bullae and ecchymoses occurred 1 week later, and the prednisone was increased to 60 mg. daily. Two days later the patient complained of severe left-sided headache, and lapsed into coma with a left third-nerve palsy and right-sided hemiparesis (Weber syndrome), and died the next day. Permission for autopsy was refused. The observed course in this patient was 15 months.

Case 3

M.T., a Bantu female aged 37, was seen in February 1955 complaining of persistent spontaneous bruising for 2 weeks, and bleeding from the mouth for 1 day; 3 months before there had been an episode of epistaxis and bleeding from the mouth. Physical examination showed generalized purpura, ecchymoses, and bullae in the mouth. The bleeding time was longer than 15 minutes and the tourniquet test was negative. Haemoglobin 7·6 g. per 100 ml., leucocytes 9,500 per c.mm. (eosinophils 0%), and platelets less than 20,000 per c.mm. Megakaryocytes were present in the marrow in normal numbers, but showed cytoplasmic hyalinization and failure to bud. A blood transfusion of 1,000 ml. was given, and cortisone, 300 mg. daily decreasing to 25 mg. daily on the 18th day. Clinical remission accompanied this therapy.

In March and in April 1955, there were recurrent episodes of purpura with platelets 20,000 and 25,000 per c.mm. respectively. The patient was seen again in August 1955 when the platelets had increased in numbers on the smears.

A further episode of purpura occurred in November 1955, and the patient was readmitted. Bleeding time longer than 15 minutes, tourniquet test positive; platelets 35,000 per c.mm. 'L.E. cells' were not detected. Spontaneous clinical remission

followed in 1 week, but thrombocytopenia persisted. In December 1955 platelets were scanty on the smears.

Re-examination in November 1956 showed haemoglobin 10·9 g. per 100 ml., platelets 40,000 per c.mm., 'L.E. cells' negative, and the spleen not palpable. There had been no further bleeding since the previous November. Admission with a view to splenectomy was refused. The course in this patient has so far been 2 years.

Case 4

E.K., a Bantu female aged 15, was admitted in July 1955 for uncontrolled epistaxis. There had been recurrent epistaxis over the previous 7 years. Physical examination was negative. Haemoglobin 6·0 g. per 100 ml., leucocytes 13,800 per c.mm. (eosinophils 138 per c.mm.), and platelets less than 25,000 per c.mm. Megakaryocytes were present in the marrow in increased numbers, but showed cytoplasmic hyalinization, 'lymphoid' forms, and failure to bud. 'L.E. cells' were not detected. Over the following 6 weeks there were repeated epistaxes requiring transfusion of 6 pints of blood. Two weeks after admission a large bulla was noted on the tongue. Splenectomy was refused.

In June 1956, platelets were less than 25,000 per c.mm. and 'L.E. cells' negative. There had been minimal epistaxis over the previous 9 months. In November 1956, platelets were 25,000 per c.mm. and 'L.E. cells' negative. Her symptoms had remained minimal, and she again refused splenectomy. The course in this patient has so far been 8½ years, with an observed course of 18 months.

Case 5

G.S., a Bantu male aged 30, was admitted to hospital in November 1954 with recurrent epistaxes for 1 month. He had had a haemorrhagic bullous eruption of the skin over the forehead 1 year before. Examination showed purpura but no bullae in the mouth. The spleen was not palpable. Blood count showed haemoglobin 9·0 g. per 100 ml., and platelets less than 20,000 per c.mm. 'L.E. cells' were not detected. The marrow showed numerous megakaryocytes but no evidence of platelet formation; hyaline and 'lymphoid' forms were noted. In spite of adequate ACTH and cortisone therapy, epistaxis, purpura and thrombocytopenia persisted during the following 2 months. Splenectomy was refused.

In May 1956, 15 months later, the patient presented in outpatients complaining of headache, and only on direct questioning about the scars on his forehead, was the previous history elicited. He had remained relatively well in the interim, but with occasional epistaxis. Examination showed a few petechiae, but there were no lesions in the mouth. Blood count showed haemoglobin 16·0 g. per 100 ml. and platelets less than 20,000 per c.mm. Bleeding time was longer than 15 minutes. 'L.E. cells' were not detected. The patient was treated with 'adrenosem' (carbayochrome salicylate) followed by ACTH, with no effect on platelets or bleeding time, over a period of 1 month. In view of this and the previous episode, he was submitted to splenectomy in June 1956, whilst still in a thrombocytopenic phase. The operation was uneventful; 4 hours after the operation bleeding time was 4 minutes and platelets returned to normal after 48 hours. The patient is well, with platelets 300,000 per c.mm., 18 months after the operation.

Case 6

D.N., a Bantu child aged 3 years, was admitted to hospital in December 1954 with a diagnosis of malnutrition. After 1 week he developed generalized purpura; he was on no treatment other than ward diet at the time. Blood count showed haemoglobin 12·0 g. per 100 ml., leucocytes 21,000 per c.mm. (eosinophils 210 per c.mm.) and platelets less than 20,000 per c.mm. Bleeding time 13 minutes. The marrow contained normal numbers of megakaryocytes, with failure to bud and the presence of forms showing cytoplasmic hyalinization.

The child remained in hospital for 7 months, during which time there was frequent epistaxis and occasional haematemesis and malaena, necessitating blood transfusion. There was no clinical response to steroid therapy and he remained thrombocytopenic throughout. In June 1955 the marrow still showed megakaryocytic abnormalities, with failure of platelet formation. Nutrition was normal after about 2 months.

In November 1956 the child was re-examined. He had experienced episodes of epistaxis since discharge from hospital. There was no purpura and no splenomegaly, but he was still thrombocytopenic (platelets less than 20,000 per c.mm.). Splenectomy was refused.

The clinical course in this case so far has been 2 years.

Case 7

K.V., a Bantu child aged 1 year, was seen in February 1956 with purpura of 3 days' duration. There was no previous bleeding and no history of drugs. No bullae were seen in the mouth, and there was no splenomegaly, but the child was febrile. Blood count showed haemoglobin 8·4 g. per 100 ml., leucocytes 7,500 per c.mm. (eosinophils 75 per cu.mm.), and platelets 25,000 per c.mm. The marrow showed megakaryocytes in normal numbers, but poor smears rendered any opinion on morphology impossible.

Blood culture taken 3 days after admission yielded *B. typhosus*, and the patient was put on to chloramphenicol; splenomegaly was noted at the time. There was clinical improvement and purpura diminished, but thrombocytopenia persisted. Three weeks after admission, with the child clinically recovered from the typhoid and no longer on chloramphenicol, there was exacerbation of purpura with platelets less than 20,000 per c.mm. Fresh episodes of purpura with thrombocytopenia persisted for another month, and the child was eventually discharged at the beginning of April 1956, still thrombocytopenic.

Three weeks later (24 April) he was readmitted with fresh purpura, and both liver and spleen were palpable. Blood count showed haemoblogin 9·8 g. per 100 ml., leucocytes 9,600 per c.mm. (eosinophils ·0%), and platelets less than 25,000 per c.mm. Six days later, with no further purpura, platelets were reappearing in the peripheral blood, and had reached 150,000 per c.mm. on discharge on 9 May.

The child was seen again on 30 October, 5½ months later, with generalized purpura. The spleen was palpable 2 finger-breadths below the costal margin. Blood count showed haemoglobin 8·4 g. per 100 ml., leucocytes 10,800 per c.mm. (eosinophils 324 per c.mm.) and platelets 40,000 per c.mm. The marrow contained numerous megakaryocytes but these failed to show any platelet budding. No further bleeding occurred in hospital and the child was discharged; splenectomy was refused.

The observed course in this child has been 10 months.

DISCUSSION

The criteria of either observing a case for over 6 months without remission or in at least 2 attacks with persistent thrombocytopenia in spite of clinical remission, as used here to establish the diagnosis of chronicity, are probably too strict; however, a history of bleeding or bruising easily or, for example, epistaxis in childhood in a case of idiopathic thrombocytopenia is not considered necessarily indicative of chronicity. For these reasons it is felt that a case must be observed for a sufficiently long period or in repeated episodes, before being labelled chronic; the cases of Lewis and Lurie[5] based on previous history or an unstated observed course, cannot be accepted as proved examples of chronic idiopathic thrombocytopenia in the Bantu.

The limited experience with chronic cases presented here does not warrant any conclusions; the cases are, however, very similar to the large group of American Whites described by Hirsh and Dameshek.[6] Of the 7 patients, 2 were children and 5 adults—1 male and 4 females. Clinical remissions in spite of persistent thrombocytopenia are common. In 3 cases, bullae in the mouth were noted accompanying relapse. The platelet count varies, being very low in relapse, and often rising, but not to normal levels, during remission. Eosinophilia is not a feature. The marrow picture is generally similar to that in the acute form, except that megakaryocytes are here more likely to be present in greater than normal numbers. Clinical remission of the first observed attack in 2 cases occurred with the administration of cortisone, but in 4 cases, 2 in the first and 2 in their second observed attacks full

dosage of cortisone was valueless in controlling bleeding. Clinical remission in one case in her first attack accompanied the administration of ACTH.

Of the 5 surviving cases splenectomy has been performed with cure in one case, and has been refused in the other 4. The two deaths were probably due to cerebral haemorrhage. It is worthwhile re-emphasising that the development of severe headache in an uncontrolled chronic idiopathic thrombocytopenia after the age of 30 is an indication for urgent splenectomy. The few cases here reported do not allow any dogmatism, but it is suggested that should a chronic case fail to respond to massive steroid therapy in a limited period, then, depending on the clinical course, splenectomy should be considered.

Thrombocytopenia associated with pregnancy is a problem of considerable practical importance. Of the acute cases 2 were 26 and 32 weeks pregnant respectively; they were discharged in remission and were not delivered in hospital. In 2 chronic cases 3 pregnancies were followed by exacerbation of the clinical picture, with resultant death of the patient in both cases. In the pregnant chronic case splenectomy should certainly be performed in the first 5 months. The 3 pregnancies resulted in live infants who showed no purpura, and delivery was not accompanied by any excessive haemorrhage.

SUMMARY

Seven cases of chronic idiopathic thrombocytopenia in the Bantu are described. The disease appears to behave no differently from that in the European with regard to clinical and haematological features. The response to steroid therapy in relapse is unpredictable. Pregnancy is often followed by severe relapse in this group.

We wish to thank Prof. J. F. Murray and Dr. K. J. Keeley for encouragement and criticism; the physicians and pediatricians at Baragwanath Hospital, Johannesburg, for their cooperation and permission to publish the cases which were under their care; Miss J. Dwolatsky, social worker, Baragwanath Hospital, for her invaluable aid in the follow-up of cases; and Mr. Haywood of the Records Department, Baragwanath Hospital, for his help.

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SOME REFLECTIONS ON MEDICAL RESEARCH AND ACADEMIC ORGANIZATION*

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In the light of contemporary progress in the medical sciences one feels the need to reflect more deeply upon the scope and aims of modern research. The individualistic or narrow approach which we have adopted in the past to the study of clinical problems was largely sterile in that it was confined to the parts thereof as though they were isolated from, and independent of, one another. The emergence of the integralistic approach which took cognizance of the interdependence of factors and of the dynamic interaction between them was thus inevitable. This approach is, happily, dominating medical thought today.

The two approaches do not cancel one another out, for both are necessary for an exhaustive evaluation of the total or holistic clinical reality. In effect, research possesses a dual nature: it must take the abstract form of evaluating the data of purely clinical studies, whether conducted by general practitioner or specialist, and it must take the strictly empirical form of experimental laboratory investigations. The final process of evaluation consists in the correlation of the data established by the two approaches. The wisdom of Claude Bernard's dictum in this regard has lost nothing of its old force: 'Entertain theories, but be not entertained by them: wear the cloak of imagination, but remove it in the laboratory.' Medicine owes a lasting debt to the philosophical approach for its correct conception of a broader understanding of aetiology and I was aware of this debt when in 1956 I was awarded the honour and the privilege of being made the Eli Lilly Research and Clinical Fellow. This senior South African Fellowship provided me with the golden opportunity of spending 11 months in the United States and of working in the surgical clinics of Boston, during most of 1957.

Boston

This New England city is the modern Mecca of medicine, and I was privileged to be stationed in the Massachusetts General Hospital, where the greatest traditions of American medicine have been developed and maintained for the past 140 years.

Under the inspiring leadership of Professors Edward D. Churchill, Francis D. Moore and J. E. Dunphy surgical training has donned a new character. The calibre of senior residents completing their training in the Massachusetts General Hospital, for example, is such that many are occupying the more important posts throughout the United States.

The research activities of the Massachusetts General Hospital, one of the affiliated hospitals of the Harvard Medical School, are staggering by South African standards. It is difficult to appreciate that over 2½ million dollars (£800,000) are spent every year on research projects, and that with the new Warren Building there are probably over 200 research laboratories in a 900-bedded private hospital. Important researches are being conducted in allergy, arthritis, cancer, cardiac biochemistry, infectious disease, and cardiac, endocrine, metabolic, gastro-intestinal, haematological, neurological, dermatological and peripheral vascular disorders; neurosurgical, orthopaedic and gynaecological pathology; adult and child psychiatry; surgical metabolism; intravenous fat administration; wound and tissue healing; anaesthesia and radiology. This is a formidable list for any single institution and it is by no means complete.

In the Surgical Division research is not a *sine qua non* for the progress of a resident or registrar, but the opportunities are such that nearly every young trainee and even mature surgeon feels inspired to participate in the investigation of current problems. Richard Varco[1] of Minneapolis said: 'the prime objective to strive for in the training of a surgeon is the cultivation of a flexible mind. The prevailing tendency of some training programmes is to destroy malleability of intellect. A young surgeon's reason becomes a captive dogma, aphorisms, arbitrary precepts, and the unthinking invocation of tradition—riddled and antiquated practices. He is deformed into conformity. This emphasis on conformity is inevitably purchased at the price of stereotyping the man's thinking. The spark of creativity is rarely kindled within the conformist. Therefore, those who insist on utter conventionality in their men, without regard to the individual's latent talents, waste a creative mind. Those places where these techniques are obligatory then become citadels of mediocrity inhabited by hypnotized incense-swingers . . . . We need more enthusiastic, capable, deeply motivated young men in research. They weave the threads of evidence into the warp of reason and the woof of truth to create knowledge as a fabric. In those institutions where research laboratories remain empty, or largely deserted, the training programme also will have a hollow ring. Those in command who are responsible for this development have lost faith in the great research opportunity available to surgeons . . . . If new appointments or promotions are unavailable to a non-conformist and enthusiastic worker and he is therewith in effect rejected, we surely add one more link to the chain of events shackling man's progress. In arriving at this decision to advance or to by-pass this man, it should be recalled that great expectations are rarely realized by anybody comfortably settled on the cushions of conformity.'

* This article was written in January 1958 and sent for publication on 7 March.

At the Massachusetts General Hospital, Boston, where I arrived in December 1956, Prof. Edward D. Churchill, Head of the Department of Surgery, who advised various lines of approach in arranging a research programme, and I joined forces with Dr. William R. Waddell, a surgeon on the staff of Harvard and the Massachusetts General Hospital. Dr. Waddell had a variety of absorbing interests and he had made noteworthy contributions in surgical physiology, particularly in the fields of fat metabolism, intravenous fat administration, atherosclerosis, gastric secretions, and the scientific evaluation of various operative procedures for duodenal ulceration. The programme finally chosen was to investigate the diverse factors related to the production of hydrochloric acid in the stomach, in which I had the facilities of a biochemical laboratory and chemist, animal operating and clinical patients.

Gastric Research

In this I had the advantage of over 2 years' previous experience in gastric and duodenal pouch work and investigation. My programme involved a great amount of study of some small part of the past literature on the subject of gastric physiology—I found that over ten thousand articles had been written. A wide field of research had to be covered and re-evaluation of past data had to be made in the light of the work of Selye *et al*[2] on stress and the interrelationships of the hypothalamic-pituitary-adrenal-gastric axis. The experiments on gastric pouches in dogs were illuminating in this respect, particularly on certain aspects of the effect of the adrenal glands on post-operative gastric acid secretions. I hope to continue the experiments in Johannesburg. Simultaneously with the animal experiments, gastric analyses were almost daily carried out on duodenal ulcer patients, mostly Dr. Waddell's.

Of the meticulous routine pre-and post-operative evaluation of gastric function employed by Dr. Waddell the most interesting operation was the Waddell procedure, viz. antral exclusion, partial sleeve resection of the stomach, and vagotomy. Up to date the results have been very encouraging when compared with the former three-quarter gastrectomy either ante-colic or retro-colic-gastro-jejunostomy.

The next phase in our researches consisted in tests on normal volunteer subjects with intravenous administration of norepinephrine and its effect on gastric secretions. This work occupied about 4 months; its results, in epitome, were that under the influence of norepinephrine the resultant vasoconstriction inhibited gastric secretion during basal states and after stimulation by peptone broth, histamine, and insulin hypoglycemia. The principal effect of this treatment was reduction of the volume of gastric juice. To my knowledge, this was the first occasion when such trials were conducted on human subjects.

Other Investigations

Besides my work in this and other gastro-enterological problems, I had the opportunity of attending vascular clinics at the Massachusetts General Hospital and the Peter Bent Brigham Hospital, Boston, and assisting for a time in cardio-vascular research with special reference to artificial heart-pump oxygenators. A third and old interest was the treatment of acute renal insufficiency by means of the artificial kidney. Dr. John Merrill, an authority on renal insufficiency, was at the Peter Bent Brigham Hospital, Boston, likewise Professor Francis Moore, a well-known surgical physiologist.

In two 3-week surgical tours undertaken to observe different research projects and to exchange ideas on the gastric physiology research we were engaged in in Boston, I visited the Johns Hopkins Hospital, Baltimore, where the emphasis was on heart-lung machines, and external pace-makers; and also Houston, where arterial graft surgery is of a very high standard. In Cleveland I spent a few days in Prof. Claude Beck's laboratory, where I participated in some experiments on coronary flow. I also had the opportunity of seeing Dr. Kolff and observing several artificial kidney dialyses; and discussing with Drs. Crile and Turnbull problems connected with gastro-intestinal carcinomata and inflammatory conditions.

Other places I subsequently visited included (1) Prof. Dragstedt's Department in Chicago, probably the most prolific gastric physiology laboratory in America; (2) Professor Wangensteen's Department in Minneapolis, where considerable gastric and cardiac research (under Dr. Lillehei) has been going on for many years; (3) the Mayo Clinic, Rochester, where I met Dr. John Kirklin, famous for his open heart operations; (4) Professor Coller's Department in Ann Arbor, Michigan, where Dr. Iob and Dr. Streeten are engaged in important metabolic researches with particular reference to the adrenal glands and gastric secretions; and (5) the Cancer Memorial Hospital, New York, where many interesting gastro-intestinal metabolic problems were being investigated under the directorship of Drs. Randall, Pack and Roberts.

My impressions were that, though a great deal of excellent laboratory and technical work on gastric problems which had been so intensively carried out over a period of years was being inadequately coordinated, yet in the light of our present more integrated knowledge of the hypothalamic-pituitary-adrenal axis, it is nevertheless assuming greater significance.

APPLICATION TO SOUTH AFRICA

It is only in recent years that surgery departments in our South African medical schools have become aware of the importance of progressive research and of the need of incorporating it in their training programmes.

In the United States, when mature and experienced professional men and women are granted funds for research work, they are also granted a basic personal allowance of about £1,000 ($3,000) a year, in addition, that is, to their ordinary academic or private earnings. In South Africa the Council for Scientific and Industrial Research do not permit a personal stipend to the person organizing and directing research. I consider this a serious defect in our research organization: too many of our research workers develop a certain cynicism in regard to the poverty of their prospects. Sooner or later they are lost to the universities, and the loss is always an irreparable one. The greatness and progress of a country can be judged today by the funds the Government devotes to scientific research. The USSR, the UK, and Germany are forging ahead and are becoming serious competitors to the USA, for that very reason. In the USA medical research is being subsidized at the rate of £1,000,000 per working day! The Union of South Africa, partly because of its meagre resources, and partly because of its lack of vision, is staggering along with an occasional discovery. It may be said that in South Africa 'full many" a research worker 'is born to blush unseen and waste his sweetness on the desert air'. The frustrations are due not only to lack of finance but also to lack of departmental heads experienced in research in basic science and free from old-fashioned concepts of training. I could name scores of young medical research workers who have been forced out of academic life because of lack of opportunities and financial means, or because just promotions are not made and the total qualities of a candidate not evaluated. In some American universities great stress is laid by selection committees on the number of publications made, and not on quality; which is probably why hundreds of contributions to modern medical literature are so unrewarding to a discriminating student. The true hall-marks of success include leadership, imagination, fearlessness in the face of pseudo-sanctimoniousness, and practical contributions to departmental administration and research.

I would say, further, that clinical acumen, however cunning, and clinical experience, however hoary, do not signify a knowledge of modern scientific methodology, and are not sufficient for leadership in the contemporary world of medical thought. Scientific methodology implies not only the identification of factors in a causal continuum and the recognition of their interdependence, as Dr. Louis Freed[3] would say, but also that the individual research worker should accept the necessity of serving as a member of an integrated research group. Any other approach must lead to corporate chaos, to personal suffering, and to inter-personal conflict.

Thus, in the light of the principles of scientific methodology, it ought not to be possible for a head of a senior clinical department to make important public statements without prior consultation with senior staff members. Staff consultations should be regularly held to discuss important matters; and when once a decision is made by the majority, it should be implemented without the possibility of its being rescinded through the caprice of a head of a department, or the dean of a faculty when a report is made to the senate. Such an approach makes for harmonious cooperation on the part of the members of a staff, for it removes the grounds of suspicion and distrust. A good leader is one who stimulates in his subordinates an enthusiasm for creative endeavour, and sustains the enthusiasm by constantly indicating the direction which progressive research should take. A good leader, too, will espouse the cause of research with convincing dignity. Such leaders are needed at the present hour.

It is impossible to conduct sound surgical research without adequate facilities for the accommodation of research animals. A modern surgical department should possess adequate facilities for the short-term and long-term housing of at least 80-100 large animals. In the United States many teaching hospitals have animal 'farms' or colonies in their basements or roofs which are large enough to house 100 dogs comfortably. Failure to appreciate this basic need has held back open cardiac surgery in South Africa by at least 5 years, and it seems that we are destined to struggle on in this way for years to come, to the detriment of the South African public. In Johannesburg we are desperately short of facilities. We need technical staff; we need biochemists; we need machinery; and we need more clinical buildings and laboratory accommodation. In the departments of medicine I visited in the United States, I found that separate and independent authority was vested in the sub-departments of Gastro-enterology, Dermatology, Endocrinology, and Metabolism and Cardiology, all with their own experimental facilities. It is this emancipation from an over-all medical dictatorship that spelt great progress in these fields in the past.

Where the employment of senior full-time personnel is the rule, there can be no place for professorial dictatorship on the Prussian model, with its apotheosis of tyranny and reaction. This system, ever ready to apply its constricting coils in a department staffed by weak or very junior personalities, is to be guarded against.

Even our senior teaching hospitals and universities refuse to improve the lot of full-time academic trained men for fear of hurting the feelings of local medical associations, who should have no say in their administration. Recently a large hospital sought the opinion of a Branch of the Medical Association of South Africa regarding a request made for permission for full-time professors to engage in private consultative practice. This permission was refused. It is extremely arguable whether the provincial authorities or the universities should, in this particular instance, have yielded to the guidance of a body which hardly represents the senior full-time academic staff of our medical schools. In nearly all progressive overseas teaching hospitals all senior full-time medical staff—and not only professors—are permitted a limited amount of private work in order to augment their shockingly inadequate salaries. This system is not abused overseas, and there is no reason to suppose that it would be abused in South Africa, where the standard of honour among medical men is not less. The advancement of our university clinical departments depends upon the self-sacrificing labours of full-time research workers and clinicians who have spent long years in hospital wards as well as in the fields of pedagogy and administration. To force men with such mature knowledge and ripe experience out of the academic cloisters of our medical schools and teaching hospitals is virtually to invite mediocrity, retrogression or disaster for the students who are to be the doctors of tomorrow. The assurance to the full-time members of the medical staff of our medical schools of relief from the spectre of economic insecurity is the only way of averting this disaster.

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EXTRAORDINARY GENERAL MEETING OF THE MEDICAL ASSOCIATION OF SOUTH AFRICA

Following are the minutes of an Extraordinary General meeting of the Medical Association of South Africa held at Medical House, 1 Wale Street, Cape Town on 9 April, 1958 at 3 p.m.:

Present: Mr. T. Schrire, Dr. L. M. Marchand, Dr. T. Shadick Higgins and Dr. A. H. Tonkin (Secretary).

It was proposed by Dr. Tonkin and seconded by Dr. Marchand that Mr. Schrire be Chairman.

Mr. Schrire declared the Meeting to be properly constituted and asked the Secretary to read the proposed amendment to Article 9 which had been published for general information in the *Journal* of 8 March, 1958.

The Secretary read the proposed amendment to Article 9 as follows:

I. That *Article* 9 be and is hereby cancelled and deleted and that the following new Article 9 be and is hereby substituted therefor:

'9 (*a*) The Council (i.e. the Federal Council of the Association referred to and defined in Article 1) shall have power on the representation of any Division or Branch and after due enquiry by such Division or Branch either through their respective Ethical Committees or, failing such Committee in the case of any particular Division or Branch, through the relevant Executive Committee (in the case of a Division) or through the relevant Branch Council (in the case of a Branch), to expel from membership of the Association any Member of such Division or Branch (as the case may be) whose conduct shall be held by the first aforesaid Council to have been such as to render him liable to expulsion under paragraph (*d*) of the last preceding Article; provided

(i) that before passing any resolution expelling such Member the said first aforesaid Council shall have satisfied itself that the Member concerned had, by registered prepaid letter, directed to his last known address, received adequate notice of the allegations made against him and of the time and place of the enquiry held in regard thereto by the relevant Ethical Committee, Executive Committee or Branch Council, as the case may be, and further that he had been given an opportunity of defending himself before such Ethical Committee, Executive Committee or Branch Council, as the case may be;

(ii) that the Member concerned has by further registered prepaid letter directed to him as aforesaid at least 28 days before the date of the meeting of the first aforesaid Council, been notified in writing of the representation made by the said relevant Ethical Committee, Executive Committee or Branch Council, as the case may be, and has been invited to submit by registered prepaid letter directed to the Secretary of the Association, at the latest 14 days before the said date of the meeting of the first aforesaid Council, for its consideration, such counter-representations in writing in his defence as he may wish to make; and further

(iii) that in case in his aforesaid counter-representation he has made a request to that effect, he has been given an opportunity of being heard in person in his own defence by the said first aforesaid Council.

9(*b*) A majority of two-thirds of those present and voting at a meeting of the first aforesaid Council shall be required for the purpose of exercising the powers of expulsion conferred on the first aforesaid Council by the preceding paragraph of this Article;

9(*c*) Any Member who has been expelled from membership in terms of this Article shall, notwithstanding that he has ceased to be a Member, be liable to pay all sums due by him to the Association, or to any Division or Branch thereof, at the time of his expulsion;

9(*d*) No Member whose conduct is under investigation or whose conduct is the subject of enquiry as aforesaid, or in regard to whom a representation as aforesaid has been made to the first aforesaid Council shall be capable of effectively resigning his membership of the Association, nor shall his membership be terminated in pursuance of any By-law until the investigation or enquiry is completed and the decision of the first aforesaid Council is made known. An investigation or enquiry shall, for the purpose of this Article be deemed to commence at the time when the matter of such investigation or enquiry is first brought officially to the notice of the Member by the Ethical Committee or Executive Committee of the relevant Division or by the Ethical Committee or Council of the relevant Branch—as the case may be—by the direction to him of the registered prepaid letter first referred to in paragraph (*a*) of this Article.'

II. That paragraph (*c*) of *Article* 10 be and is hereby cancelled and deleted and that the following new paragraph (*c*) be and is hereby substituted therefore:

'(*c*) Notwithstanding the provisions of the preceding subparagraphs (*a*) and (*b*) and without derogating from the provisions of Articles 11 and 16, no body of Members shall be finally recognised as a Division or Branch until they have complied with such conditions, if any, as regards the adoption of Rules of Organization as the Council may reasonably impose.'

III. That the following new Articles numbered respectively 29*bis*, 29*tres* and 29*quat*, shall be and are hereby added to and

inserted in Articles of Association immediately following the existing Article 29, namely:

'29*bis*. Without derogating from the general powers vested in the Council under Articles 28 and 29 and notwithstanding anything to the contrary contained or implied in any Regulation, By-law or Rule, for the better attainment of the objects of the Association in regard to the maintenance of the honour and interests of the Medical Profession or Association it shall in particular be competent for the Council to make and/or adopt Rules governing the ethical and/or professional conduct of individual Members of the Association by way of resolution carried by a majority of not less than two-thirds of the votes given thereon in the manner prescribed by the Regulations or By-laws—in the latter case in so far as not inconsistent with the former.

29*tres*. Complementary to the provisions of Article 29*bis* it shall also be competent for the Council to make and/or adopt Rules of procedure governing enquiries into complaints regarding the ethical and/or professional conduct of individual Members of the Association, by way of resolution passed by a simple majority of the votes given thereon in the manner prescribed by the Regulations or By-laws—in the latter case, in so far as not inconsistent with the former.

29*quat*. Insofar as there may at any time or from time to time be found to be any inconsistency or conflict between any relevant Rule of the Council as to ethical and/or professional conduct or as to the procedure to be followed in conducting any enquiry into complaints regarding ethical and/or professional conduct and any Rule which may have been passed or which may hereafter be passed by any Division or Branch, the Rule of the Council shall prevail, shall be of force and effect and shall be regarded as the Rule binding on all Divisions and Branches of the Association to the exclusion of any conflicting Rule of any Division or Branch.'

IV. That *Article* 30*bis* be and is hereby cancelled and deleted.

It was proposed by Dr. Marchand and seconded by Dr. Shadick Higgins that the amendments of the Articles of Association as read be adopted.

This was carried unanimously.

There being no further business the Meeting ended at 3.20 p.m.

NEW REACTOR TO BE USED FOR RADIOTHERAPY

The proved effectiveness of controlled radiation with certain types of rays as a method of therapy, notably in cancer, has raised the problem of devising methods whereby treatment can be safely given in close proximity to the massive nuclear reactors in which such rays are generated. A nuclear reactor which is expected to play an important role in this research and in the teaching programmes at American medical schools is nearing completion at the Massachusetts Institute of Technology. It is the first reactor in the US with a medical room located directly beneath the source of radiation. This will make it possible to place a patient on an operating table so as to use the beam directed through an aperture from above.

The reactor is to be fuelled with uranium-235 and in a few months it will be available for research in physics, chemistry, biology and many other fields beside medicine.

For radiation therapy, the reactor will produce neutrons and gamma rays. Radiotherapists are especially interested in the 'slow neutrons' it will send down into the medical room, which do not damage tissue as some other forms of radiation do. The neutrons have a special action on certain chemicals; for instance, a case of brain tumour will receive an injection of boron, which has the property of collecting in brain-tumour tissue, and the neutrons from the reactor will cause this boron to emit alpha particles. These particles travel only a fraction of a centimetre before they decay, but in that distance they will destroy cancerous cells in the tumour. The process, developed by a research team headed by Dr. William H. Sweet, associate professor of surgery at Harvard University medical school and visiting neurosurgeon at Massachusetts General Hospital, has already been used at the Brookhaven National Laboratory, another centre for the peaceful use of atomic energy.

During radiation the patient will be under observation by doctors who will watch him through a radiation-proof window. He will also be in telephonic communication with the doctors. Elaborate precautions are being taken to safeguard patients, doctors and the surrounding neighborhood from dangerous radiation. The device itself is a so-called 'cool' reactor, with 4,500 kg. of heavy water (deuterim oxide) circulating through it to moderate the neutrons and limit the temperature to 104°F (40°C).

The core tank containing the uranium is surrounded by shielding layers of graphite and concrete and the whole device is housed in a gas-tight steel building 22 meters in diameter.

Persons and vehicles enter the reactor building through air locks to prevent possible escape of radiation. The vehicle air lock allows trucks to haul in heavy materials for experiments or permits ambulances to take in patients for therapy. An overhead crane near the roof of the building can lift experimental materials or equipment up to 20 tons in weight. It also will lift the lead flask which will transfer spent fuel elements into the water-filled steel tank in which they will be stored pending final disposal.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

APPROVED MEDICAL AID SOCIETIES

MEDICAL AID SOCIETIES

The following list of approved medical aid societies is published for general information. Members are requested to keep this list for reference because it no longer appears in the tariff book. After each meeting of the Federal Council an up-to-date list is published in the *Journal*, including societies that have been newly approved and omitting those that have been withdrawn.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
2 June 1958

1. A.A. Mutual Medical Aid Society, P.O. Box 9595, Johannesburg.
2. Abercom Group Sick Benefit Society, P.O. Box 715, Port Elizabeth, Cape Province.
3. African Cables Medical Benefit Fund, P.O. Box 172, Vereeniging, Transvaal.
4. African Explosives Medical Aid Society, P.O. Box 1122, Johannesburg.
5. African Homes Trust Sick Fund, P.O. Box 93, Cape Town.
6. African Oxygen Limited Medical Aid Society, P.O. Box 5404, Johannesburg.
7. Afrikaanse Pers Beperk se Siekefonds, Posbus 845, Johannesburg.
8. Alex. Aitken & Carter Medical Benefit Society, P.O. Box 2636, Johannesburg.
9. Algoa Medical Aid Society, P.O. Box 369, Port Elizabeth.
10. Anglo-Alpha (Dudfield) Medical Benefit Society, Private Bag, P.O. Lichtenburg, Transvaal.

GOEDGEKEURDE MEDIESE HULPVERENIGINGS

MEDIESE HULPVERENIGINGS

Vir algemene inligting word onderstaande lys van goedgekeurde mediese hulpverenigings gepubliseer. Lede word versoek om die lys byderhand te hou want dit verskyn nie meer in die tariefboek nie. Na elke vergadering van die Federale Raad word 'n volledige lys (wat die name van pas-goedgekeurde verenigings insluit en van dié wat onttrek is weglaat) in die *Tydskrif* gepubliseer.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
2 Junie 1958

11. Argus Medical Benefit Society (Cape Argus Branch), P.O. Box 56, Cape Town.
12. Argus Medical Benefit Society (Daily News Branch), P.O. Box 1491, Durban.
13. Argus Medical Benefit Society (Star Branch), P.O. Box 1014, Johannesburg.
14. Associated Employers Medical Aid Society, P.O. Box 7462, Johannesburg.
15. A.T.I. Medical Aid Society, P.O. Box 5057, Boksburg North.
16. Atlantic Refining Company Medical Aid Society, P.O. Box 664, Cape Town.
17. Babcock and Wilcox Medical Aid Fund, P.O. Box 545, Vereeniging.
18. Bakers Ltd. European Employees' Sick Benefit Fund, P.O. Box 692, Durban.
19. Bloemfontein Municipal Employees' Medical Aid Society, P.O. Box 288, Bloemfontein.
20. Boksburg Municipal Employees' Medical Aid Fund, P.O. Box 215, Boksburg.
21. Broderick Medical Aid Society, P.O. Box 186, Vereeniging.
22. Building Societies Joint Medical Aid Fund, P.O. Box 5728, Johannesburg.
23. S. Butcher & Sons Ltd. Medical Aid Society, P.O. Box 1004, Durban.
24. Caltex Medical Aid Society (S.A.), P.O. Box 714, Cape Town.
25. Cape Portland Medical Aid Society, P.O. Box 1067, Cape Town.
26. Cape Times Medical Aid Society, P.O. Box 11, Cape Town.
27. Cape Town Municipal Employees' Association Medical Aid Society, P.O. Box 1939, Cape Town.
28. Central News Agency Ltd. Medical Benefit Society, P.O. Box 1033, Johannesburg (excluding Cape Town and suburbs, Durban municipal area, Johannesburg and Witwatersrand, and Port Elizabeth and Pretoria municipal areas).
29. Chamber of Mines Medical Aid Society, P.O. Box 809, Johannesburg.
30. Civil Service Medical Benefit Association, P.O. Box 176, Pretoria.
31. Consolidated Glassworks Limited Medical Aid and Sick Benefit Society, P.O. Box 562, Germiston.
32. Corner House Insurance Fund, P.O. Box 1056, Johannesburg.
33. Coronation Medical Aid Society, P.O. Box 1517, Durban.
34. Crookes Bros. Ltd. Medical Benefit Fund, 301 Smith Street, Durban.
35. D.F.A. Medical Benefit Society, P.O. Box 610, Kimberley.
36. Eastern Province Cement Co. Ltd. Medical Aid Society, P.O. Box 2016, Port Elizabeth.
37. E.P. Newspapers Medical Aid Society, P.O. Box 1117, Port Elizabeth.
38. Egnep Medical Aid Society, P.O. Penge, Transvaal.
39. Elwamba Medical Aid Fund, P.O. Box 42, East London.
40. Escom Cape Western Undertaking Medical Aid Society, P.O. Box 117, Cape Town.
41. Escom (N.C.U.) Medical Benefit Society, P.O. Box 30, Colenso, Natal.
42. Everite Medical Aid Society, P.O. Kliprivier, Transvaal.
43. Federated Employers' Medical Aid Society, P.O. Box 666, Johannesburg.
44. Federation of Master Printers of S.A. Medical Aid Society, P.O. Box 4465, Johannesburg.
45. Ford Medical Aid Society, P.O. Box 788, Port Elizabeth.
46. Friend Medical Aid Fund, P.O. Box 245, Bloemfontein.
47. General Mining (Associated Companies) Medical Aid Society, P.O. Box 1007, Johannesburg.
48. General Motors Medical Aid Scheme, P.O. Box 1137, Port Elizabeth.
49. Germiston Industries Medical Aid Society, 113 Pylon House, Human Street, Germiston.
50. Gledhow-Chaka's Kraal Sugar Co. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
51. Greaterman's Medical Aid Society (all branches), P.O. Box 5460, Johannesburg.
52. Hollerith Medical Aid Society, P.O. Box 7018, Johannesburg.
53. Hubert Davies Johannesburg Staff Medical Aid Society, P.O. Box 1386, Johannesburg.
54. Sir J. L. Hulett & Sons Ltd. Medical Benefit Fund, P.O. Box 248, Durban.
55. Hume Cape Medical Benefit Society, P.O. Box 7, Bellville Cape Province.
56. Hume Transvaal Medical Benefit Society, P.O. Box 204, Germiston.
57. Hunt, Leuchars & Hepburn Ltd. (Durban) Employees' Medical Benefit Fund, P.O. Box 943, Durban.
58. Hunt, Leuchars & Hepburn Ltd. (Transvaal Staff) Medical Aid Society, P.O. Box 47, Johannesburg.
59. Irvine Chapman Medical Aid Scheme, P.O. Box 316, Vereeniging.
60. Iscor Medical Benefit Fund, P.O. Box 450, Pretoria.
61. I.W.S. Medical Aid Society, P.O. Box 6946, Johannesburg.
62. J. W. Jagger & Co. Ltd. Medical Aid Society, P.O. Box 726, Cape Town.
63. Johannesburg Board of Executors' Medical Aid Society, P.O. Box 271, Johannesburg.
64. Klerksdorp Munisipale Werknemers Siektefonds, Posbus 99, Klerksdorp.
65. K. & L. Timbers Ltd. Staff Medical Aid Fund, P.O. Box 9, Elandsfontein, Transvaal.
66. Koegas Medical Aid Society, P.O. Koegasbridge, C.P.
67. Krantzberg Mines Medical Aid Society, P.O. Box 18, Omaruru, S.W.A.
68. Kroonstad Munisipale Mediese Hulpvereniging, Posbus 302, Kroonstad.
69. G. H. Langler & Co. Ltd. Medical Aid Society, P.O. Box 3762, Johannesburg.
70. Legal and General Medical Aid Society, P.O. Box 4870, Johannesburg.
71. Mail Times & Express Medical Aid Society, P.O. Box 1138, Johannesburg.
72. Marley Floor Tile Medical Aid Society, P.O. Box 67, Nigel.
73. L. H. Marthinusen Medical Aid Society, P.O. Box 64, Denver, Johannesburg.
74. Masonite Medical Aid Society, P.O. Box 57, Estcourt, Natal.
75. Metal Box Company of S.A. Ltd. Medical Aid Society, P.O. Box 7752, Johannesburg.
76. Municipal Employees' Medical Aid Society (Durban), P.O. Box 625, Durban.
77. Natal Building Society Medical Aid Fund, P.O. Box 947, Durban.
78. Natal Coal Owners' (Durban Staff) Medical Aid Society, P.O. Box 281, Durban.
79. Natal Estates Sick Fund Benefit Society, P.O. Mount Edgecombe, Natal.
80. Natal Industries Medical Aid Society, P.O. Box 1300, Durban.
81. N.T.E. Staff Medical Aid Fund, P.O. Box 39, Pietermaritzburg.
82. National Industrial Credit Corporation Medical Aid Society, P.O. Box 8296, Johannesburg.
83. National Portland Medical Aid Society, P.O. Box 21, Claremont, C.P.
84. New Consolidated Gold Fields Employees' Medical Aid Fund, P.O. Box 1167, Johannesburg.
85. Northern Assurance Co. Ltd. Medical Aid Society, P.O. Box 8615, Johannesburg.
86. Northern Medical Aid Society, P.O. Box 3437, Johannesburg.
87. Northern Rhodesia Civil Servants Medical Aid Society, P.O. Box 294, Lusaka, Northern Rhodesia.
88. Norwich Union Life Insurance Society Staff Medical and Surgical Benefit Scheme, P.O. Box 1226, Cape Town.
89. Ore & Metal Medical Aid Society, P.O. Box 3548, Johannesburg.
90. Pietermaritzburg Chamber of Industries Medical Aid Society, P.O. Box 365, Pietermaritzburg.
91. Polliack Group Medical Aid Society, P.O. Box 3008, Johannesburg.
92. Pongola Sugar Milling Co. Ltd. Medical Benefit Fund, P.O. Box 194, Durban.
93. Post Office Medical Aid Society, P.O. Box 303, Germiston.

94. Pretoria Municipal Employees' Sick Fund, P.O. Box 408, Pretoria.
95. Pretoria News Medical Benefit Society, P.O. Box 439, Pretoria.
96. Pretoria Portland Cement Co. Ltd. No. 1 Works (Hercules) Medical Aid Society, P.O. Box 405, Pretoria.
97. Pretoria Portland Cement Co. Ltd. No. 2 Works Medical Benefit Society, P.O. Box 7, Slurry, Western Transvaal.
98. Pretoria Portland Cement Co. Ltd. No. 3 Works (Jupiter) Medical Aid Society, P.O. Box 73, Cleveland, Transvaal.
99. Pretoria Portland Cement Co. Ltd. No. 4 Works Medical Aid Society, P.O. Box 26, Orkney, district Klerksdorp.
100. Printing Industry Medical Aid Society, P.O. Box 1993, Pretoria.
101. Prudential Medical Aid Scheme, P.O. Box 1097, Johannesburg.
102. Rand Public Service Medical Aid Society, P.O. Box 28, Boksburg.
103. Rand Water Board Sick Fund, P.O. Box 1127, Johannesburg.
104. Randles Bros. & Hudson Ltd. (Durban) Sick Benefit Fund, P.O. Box 1046, Durban.
105. Randles Bros. & Hudson Ltd. (Johannesburg) Employees' Sick Benefit Fund, P.O. Box 2678, Johannesburg.
106. Reckitt & Colman Medical Aid Society (S.A.), P.O. Box 1097, Cape Town.
107. 'Rennie' and 'The Consolidated' Employees' Medical Aid Fund, P.O. Box 1006, Durban.
108. Reynolds Bros. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
109. E. S. & A. Robinson (Pty.) Ltd. Medical Aid Society, P.O. Box 293, Germiston.
110. Royal-Globe Medical Aid Fund, P.O. Box 317, Cape Town.
111. Safim Medical Aid Society, P.O. Box 233, Vereeniging.
112. Safmarine Medical Aid Society, P.O. Box 2171, Cape Town.
113. Safnit Mills Medical Aid Fund, P.O. Box 11, Jeppestown, Johannesburg.
114. Santam-Sanlam Siektefonds (Alle Takke), Posbus 1, Sanlamhof, K.P.
115. Schwartz, Fine, Kane & Co. Medical Aid Society, P.O. Box 5069, Johannesburg.
116. Shell Medical Aid Society (S.A.), P.O. Box 2231, Cape Town.
117. C. G. Smith & Co. Ltd. Medical Aid Fund, 301 Smith Street, Durban.
118. S.A. Association of Municipal Employees' (S.A.A.M.E.) Medical Aid Fund, P.O. Box 62, Pretoria.
119. S.A. Breweries Medical Aid Society, P.O. Box 1099, Johannesburg.
120. S.A.K.A.V. Sick Benefit Fund, P.O. Box 33, Paarl.
121. S.A. Mutual Fire & General Insurance Co. Ltd. Staff Medical Aid Fund, P.O. Box 516, Johannesburg.
122. S.A. Mutual Life Assurance Society Staff Medical Aid Fund, P.O. Box 66, Cape Town.
123. S.A. Press Association Medical Aid Society, P.O. Box 7766, Johannesburg.
124. S.A. Teachers' Association Medical Aid Society, 12 Bellevue Road, Sea Point, C.P.
125. S.A. Torbanite (Boksburg) Medical Aid Society, P.O. Box 5083, Boksburg North.
126. South Atlantic Corporation Medical Aid Society, P.O. Box 4610, Cape Town.
127. Southern Medical Aid Society, P.O. Box 42, Cape Town.
128. Standard Brass Medical Aid Society, P.O. Box 229, Benoni.
129. Steeldale and Union Joinery Medical Aid Society, P.O. Box 1210, Johannesburg.
130. Stewarts & Lloyds Medical Benefit Fund, P.O. Box 74, Vereeniging.
131. Stuttafords Medical Aid Society, P.O. Box 69, Cape Town.
132. Sun Insurance Office Ltd. Staff Medical Aid Fund, P.O. Box 429, Johannesburg.
133. Syfret's Medical Aid Society, 24 Wale Street, Cape Town.
134. Traduna Medical Aid Fund, P.O. Box 8791, Johannesburg.
135. Transvaal Corundum Associated Asbestos Medical Aid Society, P.O. Box 72, Pietersburg, Transvaal.
136. Transvaal Society of Accountants Medical Aid Fund, P.O. Box 2995, Johannesburg.
137. U.L.A. Medical Aid Society, P.O. Box 4589, Johannesburg.
138. Umzimkulu Sugar Co. Ltd. Medical Aid Fund, P.O. Box 43, Durban.
139. United Banks' Medical Aid Society, P.O. Box 1242, Cape Town.
140. United Building Society Medical Aid Fund, P.O. Box 7735, Johannesburg.
141. University of the Witwatersrand (Johannesburg) Staff Medical Aid Fund, Milner Park, Johannesburg.
142. Vacuum Medical Aid Society, P.O. Box 35, Cape Town.
143. Village Board of Management of Welkom Medical Aid Society, P.O. Box 708, Welkom, O.F.S.
144. Wright Boag & Head Wrightson Sick Benefit Fund, P.O. Box 183, Benoni.
145. Yorkshire Medical Aid Society, P.O. Box 2755, Johannesburg.

MEDICAL BENEFIT SOCIETIES WHICH ALLOW FREE CHOICE OF DOCTOR FOR SPECIALIST SERVICES ONLY
MEDIESE BYSTANDSVERENIGINGS WAT VRY KEUSE VAN DOKTER ALLEEN VIR SPESIALISTEDIENSTE TOELAAT

1. Anglo-Alpha Roodepoort Benefit Society, P.O. Box 15, Roodepoort, Transvaal.
2. Begbie Medical Benefit Fund, P.O. Box 192, Middelburg, Transvaal.
3. Breyten Coalfields Benefit Society, P.O. Box 6, Estantia, Transvaal.
4. Broken Hill Mine Employees' Medical Specialist Fund, P.O. Box 45, Broken Hill.
5. De Beers Consolidated Mines Limited Benefit Society, P.O. Box 616, Kimberley.
6. Durban Roodepoort Deep Ltd. Benefit Society, P.O. Box 193, Roodepoort.
7. Jagersfontein Mine Benefit Society, P.O. Box 2, Jagersfontein, O.F.S.
8. Krugersdorp Municipal Employees' Medical Benefit Society, P.O. Box 101, Krugersdorp.
9. Northern Rhodesia Mine Employees Medical Specialist Fund, P.O. Box 134, Kitwe, Northern Rhodesia.
10. Public Utility Transport Corporation Sick Fund, P.O. Box 9571, Johannesburg.
11. Randfontein Estates Employees' Sick Benefit Society, P.O. Box 37, Randfontein.
12. Roodepoort-Maraisburg Municipal Employees' Association Sick Benefit Society, P.O. Box 217, Roodepoort.
13. Roodepoort-Maraisburg Non-Scheduled Mines' and Industries' Benefit Society, P.O. Box 225, Roodepoort.
14. Rosherville-Maraisburg Benefit Society, P.O. Box 99, Cleveland, Johannesburg.
15. Sasol Medical Benefit Society, P.O. Box 80, Sasolburg.
16. Simmer Pan Medical Benefit Society, P.O. Box 103, Germiston.
17. Springs Mines Benefit Society, P.O. Box 54, Springs.
18. Transvaal Jewellers' & Goldsmiths' Sick Benefit Fund, P.O. Box 8530, Johannesburg.
19. Witbank Coalfields Benefit Society, P.O. Box 26, Witbank.
20. Witbank Power Station Medical Benefit Society, P.O. Box 197, Witbank.

ASSOCIATION NEWS : VERENIGINGSNUUS

The following matters of general interest were discussed at a meeting of the Branch Council of the Cape Western Branch held at Cape Town on 23 May 1958:

Availability of Tetanus Toxoid. The sub-committee previously appointed to investigate the availability of tetanus toxoid presented its recommendations. Representations are to be made to

the Parliamentary Committee of Federal Council in favour of the general use of the triple antigen. It was stressed, however, that its use should be withheld during periods of high poliomyelitis incidence, when infants should be immunized only against diphtheria. For these occasions, or for cases where the triple antigen is not used for some other reason, representations are to be made recommending that the S.A. Institute for Medical Research should have tetanus toxoid alone readily available. It is recommended that the Parliamentary Committee should approach the Union Health Department for an amendment of Section 50 (1) (*h*) of the Public Health Act enabling an adequate supply of tetanus toxoid to be made available to local authorities.

Southern Medical Aid Society. A letter in which this Society indicated that detailed accounts (stating nature of illness) would replace claim forms in respect of services rendered to members, was laid before Council. This suggestion was welcomed by the Branch Council as being convenient for practitioners. It is pointed out that other medical aid societies have followed this course in the past.

Social Function. The Social Committee of the Branch reported on their arrangements for this year's function, which will take the form of a dinner on the night of 4 July at Arthur's Seat Hotel, Sea Point. Full details will in due course be circulated to members.

Refusal of Medical Aid Society to meet Account rendered late. This had been discussed at Branch Council and its Executive Committee on several occasions recently, and it was now felt that the attention of members should be called to the matter. Payment of a doctor's account had been refused by a medical aid society on the grounds that it had been rendered after the 6 months specified in the general rules covering the tariff. Even though the amount was a very small one, the Society has stood its ground as a matter of principle in spite of personal representations made on behalf of the Branch. Members are earnestly advised not to delay rendering accounts to medical aid societies.

Requests by Insurance Companies for Medical Findings on Patients seen at Hospitals. A representation has been made to the Branch Council concerning a recent tendency on the part of Insurance Companies to ask for particulars from the folders of patients attending Provincial hospitals. It was pointed out that this was against the interests of practitioners engaged in private practice, to whom prospective insurance applicants are normally referred by the Insurance Company concerned for a medical opinion, and who receive a fee from the Insurance Company for this service.

Problem of Alcoholism. The Branch Council discussed the suggestions of 3 members who had recently returned from a conference on Alcoholism in Johannesburg, where they had had an opportunity of seeing over some of the local institutions, and had come back feeling very strongly that Cape Town was not playing its part fully in dealing with alcoholism. They were anxious that a meeting should be arranged under the auspices of the Medical Association to discuss the problem and, in spite of the fact that as recently as last year the Branch had a symposium on alcoholism, the Council felt that these members should be given an opportunity of passing on to the Branch the benefit of their recent experiences in Johannesburg. To this end a special meeting will be arranged in due course.

Campbell Cup Golf Trophy. The competition was played at Worcester on 19 April. Drs. I. L. Bradfield and G. de V. Theron tied for first place, and the cup was awarded to Dr. Bradfield for the better score on the even holes. Dr. Theron was congratulated for giving the Western Province Amateur-ex-Champion such a good run for the trophy.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Arnold Palley, M.B., Ch.B., D.C.H., Barrister-at-law, formerly of Cape Town, has been elected to the Southern Rhodesian parliament for the Greendale constituency.

* * *

National Cancer Association of South Africa. The Annual General Meeting which had been advertised for an earlier date was postponed and will now be held on Thursday 26 June at Pharmacy House, 80 Jorissen Street, Johannesburg at 4.30 p.m.

* * *

Mr. C. A. R. Schulenberg, M.D., M.Ch., F.R.C.S. has returned from a study tour in Europe and has resumed practice at 50 van Riebeeck Medical Buildings, 295 Schoeman Street, Pretoria. *Dr. C. A. R. Schulenberg*, M.D., M.Ch., F.R.C.S. het van 'n studietoer in Europa teruggekeer en hy sit sy praktyk voort te Van Riebeeck-Mediese gebou 50, Schoemanstraat 295, Pretoria.

* * *

University of Cape Town. The affirmation of the Declaration required by candidates by the University of Cape Town before admission to the degree of M.B., Ch.B. will be made by graduands at a ceremony in the Physiology Lecture Theatre, Medical School, Observatory, Cape, at 5 p.m. on Wednesday 25 June. The graduands will be addressed by the Vice-Chancellor and Principal of the University, Mr. J. P. Duminy. Medical practitioners are invited to be present at this ceremony.

* * *

Research Forum, University of Cape Town. The next meeting of Research Forum will be held on Tuesday 17 June at 12 noon in the Large lecture theatre on A Floor, Groote Schuur Hospital, Cape Town. Speaker, Dr. C. Merskey: subject, 'Diet, Cholesterol and Coagulation'. This is the second of two meetings devoted to a report on a large cooperative study on diet, cholesterol, fibrinolysis and blood coagulation which has been made in Bantu and White males with and without overt evidence of coronary artery disease.

* * *

The South African Council for Scientific and Industrial Research invites applications for postgraduate bursaries for directed research which the Council offers for work at approved overseas institutions. Amongst the subjects for research for which bursaries may be available are the following: Enzyme chemistry, Microbiology of foods, The biological evaluation of proteins and amino-acids on humans, Psychometric methods, Biometrics, Water supply and conservation, Tissue culture, Virology, Cytology for the early identification of cancer cells, Human parasitology Coronary embolism, Hypertension, The most important causes of death and the most preventive measures for heart diseases (degenerative), Clinical psychology, Rheology. The Council will require a high standard of candidates and may make more than one award or no award in any one field of research. The bursaries will be tenable from 1 October 1958 for one year at a time and may be renewed if satisfactory reports are received on the burser's progress. The value of the bursaries will be £500 per annum for work in the UK, £600 per annum on the Continent of Europe and £800 per annum in the USA. Application may also be made for a travel grant of not more than £220. Applicants must submit programmes of research, must undertake to return to the Union of South Africa on completion of bursaries, and must make their own arrangements for entry into overseas institutions approved by the CSIR. Bursaries will be subject to the Council's regulations and applications must be on the Council's application forms R.A. 1. Forms of application are obtainable from the registrars of South African Universities or from the Secretary/Treasurer, CSIR, P.O. Box 395, Pretoria. Applications, should where possible be submitted through the Registrar to reach the Council not later than 30 June 1958.

REVIEWS OF BOOKS : BOEKRESENSIES

THE SICK AFRICAN

The Sick African. A Clinical Study. Third Edition. By Michael Gelfand, O.B.E., M.D. (Cape Town), F.R.C.P., D.P.H., D.M.R. Pp. 866. Fig. 164. 77s. 6d. Cape Town and Johannesburg: Juta & Co., Ltd. 1957.

Contents: I. The Patient. II. The Examination. III. Malaria. IV. Bilharziasis (Schistosomiasis). V. Hookworm Disease. VI. Leprosy. VII. Yaws (Framboesia

Tropica). VIII. Relapsing Fever. IX. Sleeping Sickness (Trypanosomiasis). X. Filariasis and Dracontiasis. XI. Yellow Fever and Minor Tropical Fevers. XII. Kala-Azar (Visceral Leishmaniasis). XIII. Typhoid Fever and other Causes of Prolonged Pyrexia in the Native. XIV. Undulant Fever (Abortus and Malta Fevers). XV. The Exanthemata. XVI. Veneral Diseases in the Native. XVII. Tropical Phlebitis and other Possibly related Vascular Disorders in Tropical Africa. XVIII. Plague. XIX. Amoebiasis, Dysentry and Diarrhoea in the Native. XX. Nutritional Disorders in the Native. XXI. Respiratory Affections in the Native. XXII. Diseases of the Heart. XXIII. Disorders of the Nervous System. XXIV. Affections of the Genito-Urinary System. XXV. Disorders of the Liver. XXVI. Splenomegaly in the Native. XXVII. Affections of the Bones and Joints. XXVIII. Anaemia in the Native. XXIX. Affections of the Lymphatic Glands. XXX. Disorders of the Endocrine Glands. XXXI. Disorders of the Alimentary Tract. XXXII. Malignancy. XXXIII. Common Intestinal Parasites. XXXIV. Common Skin Diseases seen in the Native. XXXV. Some Considerations of the Effects of Certain African Diseases.

This book maintains its important place as the only available textbook dealing with the diseases of Bantu races. The author's object has been to describe their diseases as seen in Rhodesia together with full reference to the literature of the subject. He has fulfilled his purpose of assisting the European-trained doctor to obtain a realistic perspective of the diagnostic and therapeutic problems of the indigenous African. He wisely corrects a common fallacy that African medicine is largely the medicine of malaria, bilharziasis, ankylostomiasis and the other common tropical diseases and while stressing the importance of general medicine throughout the book, he covers both the tropical and general aspects of each system. This edition has been enlarged and, in large measure, rewritten. Not the least valuable section is the large bibliography listing many references dealing with Bantu medicine. A most valuable chapter for those unacquainted with the Bantu is the one on the African patient. It describes his domestic, social and cultural background. He gives good descriptions of clinical differences in disease incidence, geographical distribution of conditions and the difficulties met with in African practice. One might perhaps be critical of the chapter which tells us to examine the African patient carefully and thoroughly. Although many of his hints are helpful, the objective of the chapter can surely be expressed in one sentence—'Examine your patient thoroughly'. The chapters on the nutritional disorders, the diseases of the nervous system, the anaemias and malignancy are most important contributions. Approximately half the volume deals with the more important tropical diseases of Central Africa with the emphasis on the clinical aspects. The descriptions are comprehensive, accurate and contain a wealth of clinical detail based on the wide experience and wise judgments of the author.

Doubtless, in the not too distant future, specialized works on the various sections of medicine in the Bantu will appear bringing more precise clinical and pathological descriptions of the disease patterns. Further, too, one may anticipate a composite text-book written by several contributors each an expert in a limited field.

Such works will establish more accurately the various patterns and incidence of disease in the Bantu and evaluate significant difference as compared with other groups in Africa or elsewhere.

Dr. Gelfand's conclusions are, of necessity, based on accumulation of impressions and no doubt many reputed differences of reaction in the Bantu will not be substantiated when better statistical control is possible. Nevertheless, at the present time, his contribution to this vast field is most valuable and this book will be of assistance to all medical personnel who are concerned with the practice of medicine in the Bantu peoples.

G.H.J.

ATLAS OF EYE SURGERY

Atlas of Eye Surgery. By R. Townley Paton, M.D., F.A.C.S., Herbert M. Katzin, M.D., F.A.C.S. and Daisy Stilwell (Illustrator). Pp. viii+247. 463 Illustrations. $15.00. New York—Toronto—London: McGraw-Hill Book Company, Inc. 1957.

Contents: 1. General Preparation. 2. Preliminary Consideration. 3. Anesthesia. 4. Operations for Cataract. 5. Operations for Glaucoma. 6. Operations for Retinal Detachment. 7. Corneal Transplantation. 8. Enucleation. 9. Evisceration. 10. Operations on the Extraocular Muscles. 11. Operations for Pterygium. Index of Instruments.

There have been numerous text-books on Ophthalmic Surgery. Among recent volumes, we have had some encyclopaedic, such as Spaeth; some personal techniques such as Stallard. There have also been previous Atlases on the subject, such as Arruga's. The last, while mainly concentrating on the author's personal tecniques, has attempted to be more general and therefore has had a largish amount of accompanying text. The present volume is practically all illustrations (and what wonderful ones they are!) with the running commentary reduced to a minimum. For teaching purposes obviously unbeatable.

The authors are extremely well known and Paton is the author of the authoritative 'Keratoplasty'. In an extremely well-produced volume they have given the routine management of the operations for cataract, glaucoma, retinal detachment and so forth, as performed by the authors. If one wishes to perform the straightforward, routine, safe operation or to standardize one's own technique, no better means could be found to assist. Of course one may adopt one's own modifications, for example, it is not necessary to use the Berens muscle clamp or catgut in squint surgery or to do a double iris pillar inclusion in iridencleisis, but every book cannot be encyclopaedic.

A useful point is that all the illustrations are drawn as seen by the surgeon from the position he usually adopts, namely from above and behind the patient's head. This book can be recommended to every eye surgeon, beginner or experienced.

S.L.L.

BIOLOGICAL ASPECTS OF CANCER

Biological Aspects of Cancer. By Julian Huxley, F.R.S. Pp. 156. 16s. net. George Allen & Unwin Ltd. 1958.

Contents: Preface. *A. Comparative and Genetic*. I. Introduction. II. Comparative Distribution of Neoplasms. III. Tumour Genetics. *B. Viral and Epigenetic*. I. Carcinogenic Viruses. II. Other Viruses. III. Tumours and Epigenetics. *C. Summary*. References. Index.

The publication of Dr. Julian Huxley's book on cancer as a biological problem is an event of importance to all who are engaged on research in this field.

The author spent a few years of hard labour in making himself familiar with the vast mass of literature on cancer that has appeared in the last decades, and has assessed the information obtained from it against his profound knowledge of biology in general and genetics in particular. The result of his efforts is a work containing an amazing diversity of information on many disciplines concerning cancer that was hitherto not available in a comprehensive survey by an author of high scientific standing.

It is not a book for the medical practitioner whose mind is far removed from modern biology or fundamental research on cancer. Nor is it easy to read for research workers who are not familiar with the science of genetics. In fact, the author seems to expect of the reader a scientific background almost as wide as his own. It is a stimulating book that should inspire the research worker to broaden his outlook on the many sided aspects of the cancer problem.

J.v.R.

SCHIZOPHRENIA IN OFFICE PRACTICE

Schizophrenia in Psychoanalytic Office Practice. Edited by Alfred H. Rifkin. Papers presented at the 1956 Symposium of the Society of Medical Psychoanalysts held in New York. Thirty Contributors. Pp. x+150. $4.00. New York and London: Grune & Stratton, Inc. 1957.

Contents: Contributors. Foreword. *Concepts. Special Problems:* Panel I. Transference and Related Problems. Panel II. Anxiety, Panic and Other Problems. *Treatment*.

The problem of schizophrenia has been exercising the minds of psychiatrists since the end of the last century, and at no time more than the present. Its importance is felt to be so great that one of the authors suggests that the borderline schizophrenic is 'the neurotic personality of our time'. It was therefore inevitable that psychoanalysts in all countries have been called upon to treat schizophrenics both in mental hospitals and in office practice, and as a result different groups have evolved varying theoretical concepts and techniques of treatment.

This volume is composed of seventeen papers presented by the members of a New York group. Although due consideration is given to the many etiological causes, both organic and otherwise, which have been put forward, the preface states that 'implicit in all these discussions is the premise that the development of schizophrenia is intimately related to the conditions of life'.

The first section consists of three papers on the nature of extramural schizophrenia, schizophrenic consciousness in practice, and the conceptual vicissitudes of the idea of schizophrenia, together with a summary of these concepts. Each paper is discussed by two or three contributors, and serves to illustrate how considerable are the differences in opinion.

The second section deals with the difficulties met with in the transference, and some very practical issues, such as suicide, hospitalization, and the role of the family.

The third section deals with the selection of patients, treatment of a-social attitudes, and a paper on the psychic structure and

therapy of schizophrenia. This also has a summary and a discussion.

This symposium, while offering no final answers to the understanding and treatment of schizophrenia, shows a constructive and hopeful approach and supplies some helpful ideas and suggestions.

S.A.W.

AIDS TO ANAESTHESIA

Aids to Anaesthesia. 4th Edition. By Victor Goldman, F.F.A.R.C.S., D.A., L.R.C.P., M.R.C.S. Pp. vii+359. 92 Figures. 12s. 6d. London: Baillierè, Tindall & Cox Ltd. 1957.

Contents: Preface. I. History of Anaesthesia. II. Biographies. III. The Anatomy and Physiology of Anaesthesia. IV. The Pharmacology of Anaesthetic Agents. V. General Considerations: The Stages of Anaesthesia. VI. The Examination and Preparation of the Patient. VII. Inhalation Anaesthesia. VIII. Nitrous Oxide. IX. Closed Circuit Anaesthesia or Complete Rebreathing. X. Vinyl Ether—Vinesthene—Divinyl Oxide. XI. Cyclopropane. XII. Intravenous Anaesthesia. XIII. Endotracheal Anaesthesia. XIV. Premedication. XV. Complications of Anaesthesia. XVI. Anaesthetics for Diseased Patients. XVII. Anaesthetics for Special Operations. XVIII. Muscle Relaxants. XIX Controlled Hypotension. XX. Analgesia and Anaesthesia in Midwifery. XXI. Anaesthesia for Dental Operations. XXII. Anaesthesia in the Tropics. XXIII. Local Analgesia. XXIV. Spinal Analgesia. XXV. Appendix of Useful Information. Index.

This book contains a brief outline of most aspects of anaesthesia. The information given is for the most part of an extremely elementary nature, especially those sections dealing with Anatomy, Physiology and Pharmacology. The author makes no pretence of providing detailed description of apparatus or techniques or of discussing them, but has deliberately confined himself to summarizing common practice.

The section on anaesthesia for dental operations is the fullest in the book. As one would expect from Victor Goldman who is best known in this field, it contains a fund of practical information based on vast personal experience.

As a whole, this little book should be most valuable to nursing staff engaged in theatre work, as it contains all they need to know concerning anaesthetic methods and their underlying theory. For the undergraduate, it gives a fair outline of current anaesthetic practice. The post-graduate student or the specialist anaesthetist should find the appendix of useful information a source of great joy and a real aid to his practice.

A.B.B.

DISEASES OF BONE

Bone Diseases in Medical Practice. By I. Snapper, M.D. Pp. x+229. 48 Plates. 815.00. New York and London: Grune & Stratton, Inc. 1957.

Contents: Preface. 1. Physiology of Bone. 2. Semantics of Bone Disease. 3. Osteoporosis. 4. Rickets and Osteomalacia. 5. Hypervitaminosis D. 6. Hypervitaminosis A. 7. Infantile Cortical Hyperostosis. 8. Hyperparathyroidism. 9. Renal Osteitis Fibrosa. 10. Metastatic Calcification after Medical Treatment of Peptic Ulcer. Burnett's Syndrome. 11. Boeck's Sarcoidosis. Nephrocalcinosis and Skeletal Involvement. 12. Hyperparathyroidism and Pseudohypoparathyroidism. 13. Paget's Disease of the Bone. (Osteitis Deformans). 14. Osteoporosis Circumscripta Cranii. 15. Hand-Schüller-Christian's Disease; Letterer-Siwe's Disease; Eosinophilic Granuloma. 16. Osteitis Fibrosa Disseminata (Albright) or Polyostotic Fibrous Dysplasia. 17. Leontiasis Ossea. 18. Bone Changes in Hemolytic Anemias. 19. Bone Lesions in Lymphomatous Diseases (Hodgkin's Disease, Lymphosarcoma and Reticulum Cell Sarcoma). 20. Skeletal Lesions in Leukemia and Leukemia-like Conditions. 21. Multiple Myeloma. 22. Gout. 23. Gauscher's Disease. 24. Osteopetrosis, Albers-Schönberg's Disease. Marble Bone Disease. 25. Neurofibromatosis of Bone. 26. Hypertrophic Osteoarthropathy. 27. Scleroderma. 28. Charcot Joints. Neurotrophic Osteoarthropathy. 29. Skeletal Lesions in Cancer of Breast, Prostate and Thyroid. 30. Fungus Diseases and Actinomycosis. 31. Differential Diagnosis of Bone Lesions. Index. Plates.

Dr. Snapper has been a Professor of Medicine in four Medical Schools in three Continents and has contributed a great deal of original work and thought in a number of fields of medical practice, not least diseases of bone. This book is the result of a life-time of study in this field. In it he sets out to cover bone diseases which occur in medical practice. Naturally enough the subjects closest to his field of original work receive greatest consideration, e.g. Osteomalacia and hyperparathyroidism—but he has also written very adequate simple descriptions of many of the other diseases of bone. There is little that is controversial. The book seems to be written at a level which a general practitioner might aim at achieving, and certainly contains little more than a good final year medical student might be expected to know. But what it does contain is authoritative, factual and eminently readable.

The X-rays are beautifully reproduced. The emphasis has been on the later stages of disease where the pictures are more striking, but some of the earlier changes on X-ray are also shown; more of the latter might have been included. It is a pity however that the X-ray plates have had to be collected together at the end of the book for this interferes with easy reading.

The book has other slight defects. There is a tendency to use proprietary names for drugs which may be unfamiliar to many readers, e.g. Basal-jel with small doses of Hytakerol as recommended for the treatment of Paget's disease. More careful proof reading might have eliminated a few errors. For instance 'Coomb's test' and the 'Ashly' technique for measuring red-cell survival as well as a quite incomprehensible sentence on page 36. But these are really only minor criticisms of a book which maintains as high a general standard as this one does.

What should be included in a book with the title of 'Bone Diseases in Medical Practice'? Clearly Dr. Snapper regards this as including more than metabolic bone disease. If one includes leukaemia, Hodgkins disease and gout what justification is there for omitting primary tumors on bone, many of the inflammatory diseases of bone or all the developmental diseases of bone? The author gets round this problem by ignoring it completely and since the reviewer can think of no better solution perhaps he has adopted the wisest course.

M.C.

THE CLINICAL APPLICATION OF HORMONE ASSAY

The Clinical Application of Hormone Assay. By John A. Loraine, M.B., Ph.D. (Ed.), M.R.C.P. (Ed.) with a Foreword by J. H. Gaddum, Sc.D., F.R.S. Pp. xii+368. 66 Figures. 30s. net+1s. 5d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

Contents: I. General Principles in Hormone Assay. II. Pituitary Gonadotrophins. III. Human Chorionic Gonadotrophin. IV. Thyrotrophin. V. Adrenocorticotrophin. VI. Growth Hormone. VII. Prolactin. VIII. Antidiuretic Hormone and Antidiuretic Substances. IX. Oestrogens. X. Progesterone and its Metabolites. XI. Corticosteroids. XII. 17-Ketosteroids and Androgens. XIII. Adrenaline and Noradrenaline. XIV. Insulin. Index of Authors. Subject Index.

Few publications have come at a more opportune moment. Interest in clinical endocrinology is becoming more and more widespread, and until now there has not been a 'ready reference book' to guide the clinician in the theory and practice of hormone assays: surprisingly few doctors who order these estimations have any idea exactly what they are asking for, or of the limitations of the methods used, or of the validity of the results which are taken as gospel truth by so many. The publishers have described the book as 'For experimentally-minded clinicians'—I would like to suggest 'For all doctors who order hormone estimations' as a more suitable label.

In his preface, the author says that the book was written with a three-fold purpose: to review existing methods of hormone assay, to discuss their application to clinical work, and to suggest further fields for their application. These purposes have been admirably fulfilled, and though the book is intended primarily for the endocrinologist, Dr. Loraine is to be congratulated on having put a very involved subject well within the compass of any present-day physician.

The first chapter is a succinct summary of the general principles that underlie assay procedures: in the other chapters, the hormone, or group of hormones, is discussed under the headings of—chemical nature, theories about function, methods of assay, and the possible clinical application of these methods. Reference to subject matter is greatly facilitated, and one is not compelled to wallow through the slough of pseudo-essay that characterizes so much contemporary endocrine writing. In discussing adrenal diagnostic procedures, proper emphasis is laid on the work of Norymberski *et al.*, and it is high time that these methods succeeded the estimation of the less specific 'formaldehydogenic' or 'reducing' steroids. The noteworthy chapter on pituitary gonadotrophins is of the high standard that one would have expected from the author who has made signal advances in the methods of their assay. Incidentally, many endocrinologists will be interested to own a book that has only one reference to Selye!

Messrs. Livingstone have produced the combination of excellent printing and reasonable price that we have learnt to expect of them, though they would endear themselves to Natal doctors if they could ever devise a cockroach-proof cover!

I can recommend this book to all modern physicians and clinical pathologists as a 'must', and to all prospective writers of medical textbooks as an example of how one should be written, short, clear, concise: its effect on endocrine practice will be immeasurable. Professor Gaddum says in his foreword—'No clinical endocrinologist can afford to be without it: all those that are interested in the clinical application of hormone assays will find it valuable.' There could be no better tribute than such words from a distinguished scientist.

G.D.C.

CORRESPONDENCE : BRIEWERUBRIEK

MORTALITY AMONG SOUTH AFRICAN DOCTORS

To the Editor: All the analyses in the article under this title in the *Journal* of 10 May 1958 (p. 498), are based on the total population of doctors without reference to the age distribution of the doctors at risk.

Comparisons of rates between populations should be age specific or standardized. In this case the ages of the population at risk are not known and the tentative conclusions are likely to be misleading.

Table II (percentage deaths at various age-groups) does not allow the deduction 'that only 15·35% of doctors reach the age of 75 years'. Here again the percentages depend on the numbers at risk at each age. It would be necessary to use a method such as the construction of a life table to make this kind of decision.

A. M. Adelstein

201 Eden Roc
26 Bruce Street, Hospital Hill
Johannesburg
28 May 1958

ACHALASIA OF THE CARDIA

To the Editor: I found your Editorial[1] on achalasia of the cardia of great interest but was surprised to note that the words achalasia and cardiospasm were used synonymously.

Achalasia is not a disease of the cardia but a neuromuscular dysfunction of the oesophagus as a whole. The normal oesophageal vestibular tone is unable to relax because of the absence of any stimulating peristaltic waves. The treatment is, as you state, very difficult.

In cardiospasm, on the other hand, the muscle of the vestible is contracted beyond the normal resting tone. There is no abnormality of the neuromusculature of the body of the oesophagus. The dilatation of the oesophagus which occurs is never extensive and is entirely secondary to the distal obstruction. This cardiospasm is usually the response to some irritative upper abdominal focus, e.g., peptic ulcer or biliary disease. Recovery may be spontaneous or may occur after treatment of the primary cause.

This differentiation is important as it lends itself to a better appraisal of the final results of treatment in these conditions.

W. Silber

316 Medical Centre
Heerengracht, Cape Town
6 June 1958

1. S.A. Med. J. (1958): 32, 555 (31 May).

RODENT ULCER

To the Editor: Mr. Hersch's interesting comment on my article raises several points. It is clear that he feels that these common tumours should be attacked surgically, rather than with radiotherapy, and that what is good enough for the small type (Group 1) and the complicated type (Group 3), is good enough for the ordinary average type too. I am afraid, however, that Mr. Hersch has missed my point; the object is to treat the cases with radiotherapy because an experience with 1,200 cases of rodent ulcer and about 500 cases of lip cancers has shown to our satisfaction that this treatment is safe, is usually successful, and can be done as an out-patient procedure; the horrifying necroses that are encountered elsewhere have happily not plagued our patients. The early case is excised because it is as easy to do this as to take a biopsy; the complicated case is treated with surgery because there is no alternative; but the average case is ideal for radiotherapy and we are happy to pass it on to those expert in this procedure. It is a question of what is best for the patient rather than 'out of loyalty to the radiotherapeutic members of the Combined Clinic'.

I note the difficulty Mr. Hersch has with radiotherapy, and can only say that I welcome the opportunity of paying a tribute to the quality of the radiotherapy that has become available in Cape Town since we established the Combined Clinic in 1948. Mr. Hersch is not so lucky, and it seems to me that he should bend his efforts towards improving the radiotherapy in the hospitals he frequents rather than try to recommend surgery—at best a second-rate form of therapy for cancer, and only used because we have no better available—for all and sundry. Drs. R. D. Tucker and M. B. Bennett have shown me over the years that it is possible to get excellent results in a very large proportion of cases, and, if the radiotherapists are not greedy or reckless, that sufficient cases get referred for surgical attention to keep up the interest of the surgical team.

Another point that arises is that the bulk of these patients are representative of farmers all over the world; they are conservative, superstitious and stubborn. It takes a lot to persuade them to come for medical treatment, rather than go to the quack and be destroyed, and the legend that to 'cut a cancer is to kill a patient' dies hard in our part of the country. The fact that we can record over 1,000 cases with no deaths in a long follow-up is, we submit, a very satisfactory state of affairs and a tribute to the quality of work and the persuasive powers of our radiotherapeutic staff.

T. Schrire

17 Rugby Road
Oranjezicht, Cape Town
7 June 1958

HERNIOTOMY UNDER HYPNOSIS

To the Editor: P.S., carpenter cum professional hypnotist, aged 24, was seen on 30 November 1957. He stated that, after an operation for bilateral congenital undescended testes and associated herniae in 1952, the left hernia recurred in January 1957 after a strain. A small lump appeared which enlarged gradually to the size of a tennis ball, and became painful. A large bubonocele was present, the size of half a tennis ball. His senior partner in hypnosis, Mr. M. was present, and stated that S. had been conditioned by him to have the operation under hypnosis. He told S. to 'put himself deep', and S. appeared to go into a trance. The genuineness of this was accepted without testing, and the operation was arranged.

Operation. Four days later, at 8 a.m., in the presence of 4 anaesthetists, and without any premedication, I told S. to 'put himself deep'. Mr. M. was not present, and he hesitated, stating that he was nervous in front of so many doctors. He then composed himself and said that he was now ready. The instruction was repeated along these lines—put yourself deep. You will feel no pain. Relax you muscles completely. You will try to stop any bleeding. He went into a trance as before. The skin was incised without any reaction. Bleeding was no less than usual and, though the appropriate instruction was repeated, gave the usual difficulty. Many adhesions were found, and the cord was difficult to free. The patient groaned deeply on 6-8 occasions while this was done. At one stage his eyelids fluttered and he appeared to be waking. He responded to a further instruction to 'go deep'. The skin scar was excised, the large sac was removed after ligation of the neck, and a stainless-steel mesh patch was sutured over the muscular defect with nylon. The operation lasted 1½ hours.

Observations. Sensation and reflexes were normal before the operation was started. The pulse rate was 70 per minute then, and rose during the operation to 100. The patient sweated profusely and became pale. At 10.30 a.m. his pulse was 72, and half-hourly readings thereafter were 72, 68, 68, 70, 80, 74, 72. He got up on the evening of the same day, as all signs of shock had disappeared by 10.30 p.m. No treatment was given for shock, apart from recumbency. Convalescence was uneventful, and he was discharged on the 6th day, the wound having healed well. Follow-up on 3 January 1958 was satisfactory, and again on 3 June.

M. Arnold

Medical School
University of the Witwatersrand
Johannesburg
3 June 1958